PHYTOPATHOLOGY

VOLUME 1 NUMBER 6

DECEMBER, 1911

# ROOT AND CULM INFECTIONS OF WHEAT BY SOIL FUNGI IN NORTH DAKOTA

T. D. BECKWITH

Believing that current ideas with regard to the lowered wheat production per acre in the Northwest could not be accepted as the whole cause of the deterioration in such yields, it was decided to make further examinations concerning certain additional factors which are known, at least to contribute to the result.

It has been shown by Bolley [1] that certain fungous diseases of specific character, notably Fusarium wilt of flax, may spread through the soil from a centre and thus cause a progressive diseased condition of the crop radiating from some point of original infection. Thus it was shown why soil may be "sick" for flax. Later it was shown by Selby and Manns [2] that a Fusarium causes a scab on wheat often in association with an anthracnose belonging to the Colletotricha. The two Fusaria are not the same species apparently. In the meantime the author had been demonstrating the presence of these two diseases on wheat together with certain other parasitic fungi. The same forms were found in abundance in old wheat soils which had become "worn out" according to the views of the farmers. The results of these investigations were summarized very briefly in a paper before the American Phytopathological Society in December, 1909[3]. Bolley [4] has amplified these and other results, discussing the presence of these forms on wheat and other cereals and the soil in which they grow. Also he has called attention to the need of proper methods of tilth for conservation of crops infected with these diseases. Pammel *et al*[5] have demonstrated the presence of two Helminthosporia on barley in 1910 and DeLoach[6] calls attention to Colletotrichum on cotton. Also Loew[7] calls attention to the necessity of soil conditions hygienic and sanitary to the plant in order

[1] Bolley, H. L. North Dakota Agr. Exp. Sta. Bull. 50. 1901.
[2] Selby, A. D. and Manns, T. F. Ohio Agr. Exp. Sta. Bull. 203. 1909.
[3] Beckwith, T. D. Science, N. S. 31: 798. May 20, 1910.
[4] Bolley, H. L. Science, N. S. 32: 529–541. Oct. 21, 1910.
[5] Pammel, L. H., King, Charlotte M., Bakke, A. L. Iowa Agr. Exp. Sta. Bull. 116. 1910.
[6] DeLoach, R. J. H. Georgia Agr. Exp. Sta. Bull. 85. 1909.
[7] Loew, Oscar. Porto Rico Agr. Exp. Sta. Circ. 12. 1910.

[PHYTOPATHOLOGY, for October, 1911 (1: 139–168, *pls.* XXII-XXIV) was issued October 10, 1911.]

to obtain the best results in crop production. This is but a partial list of publications during the last few years dealing with these diseases and the idea of clean soil for crop production. The writer since 1907 had been studying these forms and their occurrence on wheat and in "wheat-sick" soil.

Quantitative analyses made from soil solutions taken from soil samples from old wheat lands and from virgin soil fail to show sufficient difference in composition to warrant the assumption that deterioration in wheat yields is due entirely to depletion of plant food in the soil. Without considering carefully whether the soil toxin theory of the Bureau of Soils is to be accepted as the most potent factor in the specific sickness of old wheat soils, we determined to make a survey of certain experimental areas on the North Dakota Experiment Station farm, with a view to ascertaining what genera of certain parasitic fungi are present.

The area selected consisted of a strip divided into twenty plots, each approximately 20 x 30 feet. This strip of land has been devoted to wheat constantly for about forty years, and in consequence the yield per acre had fallen very materially. At the same time, as stated before, analyses of the soil solutions prepared by adding one part of soil to ten parts of distilled water showed a good supply of plant food. To deficiency of plant food then could not be attributed the cause of yield deterioration.

In the survey for fungi the following method was used. A soil solution was made by mixing one part of soil with ten parts of distilled water. This mixture was agitated frequently for twenty-four hours, in order that such readily soluble matter as was present in the soil might go into solution. This soil solution was then filtered through Pasteur bougies in order to clarify it, and then without further treatment, was made into a solid medium by the addition of one and one-half per cent of agar-agar. The reaction of this medium was very slightly acid, being .02% normal.

Soil samples were collected from a depth of two inches below the surface using proper precautions to guard against contamination, and a suspension made by adding one gram of the sample to 10 cc. of sterile distilled water. After two minutes agitation in order to obtain as even a mixture of the soil particles and water as possible, this suspension was plated out on the soil agar. Plates were incubated four days at 29°-30°C. Growth was very slow and much restricted on account of the small amount of nutrient material present in the medium. Fungous and bacterial colonies never grew to any size, and could be fished out without difficulty. After purifying the fungous cultures, their morphological characteristics were studied on beef agar, potato

sterile wheat straw, etc. The following genera of fungi were found: Fusarium, two species; Colletotrichum, two species; Macrosporium, Alternaria, Spicaria, Verticillium, Rhapalomyces, Cephalothecium and Helminthosporium. Of these Fusarium, Colletotrichum, Macrosporium, Alternaria and Helminthosporium are recognized pathogens of wheat and other cereals.

The next series of cultures was intended to show what genera of fungi would have spores or vegetative mycelium on or in the various parts of the wheat culm, the idea being to show, not what per cent of specific infection had taken place, but rather what potentialities for such infection might be present. The method followed was to select from each of the twenty beds at random ten wheat plants, making 200 plants from the strip. These plants were selected haphazard in order that as representative a survey as possible with the time at hand might be obtained. The method pursued for this examination was to place the specimen in a warm, moist atmosphere in order that any fungous spores present might germinate and then their growth be examined microscopically.

Germination tubes were prepared by placing a plug of absorbent cotton about one-half inch thick in the bottom of a test tube and then moistening it with a little distilled water. The tubes were then plugged and autoclaved, after which the sections of stems were introduced.

A piece of nearly mature culm of the wheat about three inches long and cut to include the first aerial node was then prepared and placed in one of the tubes. Another section of the plant was also selected, this being cut so as to include the second internode only. These various sections thus composed a double series of 200 each, one, the first aerial nodes of 200 wheat plants and the other the succeeding internodes from the same 200 plants. After one week's incubation at 29-30°C. microscopic examination was made.

The following table shows possibilities for infection found on these non-sterile internodes of the plants taken from the series of twenty plots.

| Plot Number | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | 14 | 15 | 16 | 17 | 18 | 19 | 20 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Colletotrichum sp. | 60 | 100 | 90 | 80 | 70 | 80 | 80 | 100 | 80 | 50 | 100 | 80 | 100 | 100 | 90 | 90 | 100 | 80 | 90 | 40 |
| Macrosporium sp. | 30 | 40 | 40 | 60 | 30 | 40 | 50 | 40 | 30 | 70 | 50 | 90 | 70 | 60 | 100 | 80 | 10 | 50 | 20 | 50 |
| Helminthosporium sp. | 90 | 80 | 60 | 30 | 30 | 90 | 70 | 80 | 60 | 20 | 40 | 50 | 50 | 70 | 60 | 40 | 20 | 80 | 20 | 30 |
| Cephalothecium roseum | 10 | 10 | 10 | 10 | 0 | 10 | 0 | 0 | 20 | 50 | 20 | 10 | 0 | 0 | 0 | 0 | 0 | 10 | 20 | 0 |

The following table likewise shows the percentage of nodes showing the possibilities of infection on or in the internodes.

| Plot Number | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | 14 | 15 | 16 | 17 | 18 | 19 | 20 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Colletotrichum sp. | 80 | 90 | 100 | 90 | 80 | 100 | 100 | 100 | 90 | 80 | 100 | 80 | 100 | 90 | 80 | 90 | 100 | 90 | 100 | 60 |
| Macrosporium sp. | 40 | 50 | 90 | 90 | 70 | 70 | 50 | 30 | 50 | 70 | 70 | 80 | 70 | 80 | 90 | 70 | 50 | 50 | 70 | 60 |
| Helminthosporium sp. | 100 | 80 | 60 | 40 | 40 | 90 | 60 | 70 | 50 | 50 | 40 | 20 | 40 | 100 | 90 | 70 | 80 | 60 | 70 | 40 |
| Cephalothecium roseum | 10 | 30 | 0 | 30 | 10 | 40 | 0 | 10 | 10 | 40 | 10 | 0 | 0 | 0 | 10 | 0 | 0 | 0 | 10 | 0 |

A summary of these tables appears below :

| | Nodes | Internodes |
|---|---|---|
| Colletotrichum sp. | 90 | 83 |
| Macrosporium sp. | 65 | 50.5 |
| Helminthosporium sp. | 62.5 | 58.5 |
| Cephalothecium roseum | 10.5 | 9 |

This second series thus showed that the spores or vegetative mycelium from some of the fungi which were in the soil are to be found likewise in or on the nodes and internodes of the wheat. The next step was to show whether any of these fungi were infecting the wheat plants or whether the examination had revealed merely the presence of certain fungous spores which had merely lodged on the outside of the plant without being the cause of further trouble.

It was planned to try to find out whether or not certain of these fungi were living parasitically in the wheat, thus giving rise to a pathological condition. Again, as before, for the second group there was prepared a double series consisting of 200 nodes and 200 internodes which were incubated at 30° C for 7 days. Each section of the wheat stem was placed in a test tube prepared as before. In this last series, however, the stem sections were first sterilized by treating with one per cent formaldehyde for fifty to sixty seconds and then washing in sterile distilled water to remove the formaldehyde. Such a procedure was decided to be sufficient to cause external sterilization from fungus spores without killing internal mycelium.

Following are given the results showing the infection of wheat stems during the season of 1909. The first table gives the percentages of infected internodes and the second the nodes.

It will be noticed that in the unsterile series Cephalothecium was found, while Fusarium is not noted. In the last or sterile series Fusarium often occurred while Cephalothecium was found but once or twice. This seems to show that surface sterilization was complete, for

I. INFECTED INTERNODES. 1909. STERILE

| Plot Number | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | 14 | 15 | 16 | 17 | 18 | 19 | 20 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Colletotrichum sp. | 50 | 40 | 10 | 30 | 30 | 90 | 30 | 20 | 30 | 20 | 60 | 70 | 50 | 90 | 50 | 70 | 80 | 80 | 60 | 90 |
| Fusarium sp. | 20 | 40 | 0 | 30 | 30 | 40 | 60 | 20 | 30 | 0 | 30 | 10 | 20 | 60 | 10 | 40 | 10 | 40 | 40 | 30 |
| Macrosporium sp. | 20 | 20 | 30 | 80 | 60 | 30 | 20 | 30 | 70 | 20 | 20 | 50 | 50 | 20 | 10 | 20 | 20 | 20 | 40 | 30 |
| Helminthosporium sp. | 50 | 20 | 20 | 50 | 20 | 70 | 50 | 0 | 30 | 20 | 20 | 0 | 70 | 100 | 40 | 30 | 40 | 40 | 20 | 0 |

II. INFECTED NODES. 1909. STERILE

| Plot Number | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | 14 | 15 | 16 | 17 | 18 | 19 | 20 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Colletotrichum sp. | 100 | 50 | 30 | 40 | 30 | 90 | 30 | 20 | 30 | 20 | 90 | 50 | 50 | 90 | 60 | 60 | 90 | 70 | 100 | 40 |
| Fusarium sp. | 70 | 70 | 0 | 30 | 50 | 60 | 20 | 40 | 10 | 10 | 30 | 20 | 40 | 60 | 0 | 20 | 10 | 50 | 20 | 30 |
| Macrosporium sp. | 60 | 60 | 40 | 90 | 80 | 50 | 20 | 70 | 60 | 40 | 30 | 50 | 70 | 60 | 40 | 60 | 20 | 40 | 70 | 60 |
| Helminthosporium sp. | 70 | 50 | 10 | 20 | 20 | 80 | 50 | 10 | 50 | 50 | 30 | 10 | 70 | 100 | 20 | 20 | 60 | 30 | 30 | 30 |

the saprophyte was eradicated and the less active parasitic types then had a chance to grow or to become apparent.

It is very possible to imagine that with varying climatic conditions with their necessarily associated sequent conditions, the percentage infection by these diseases may vary materially from year to year. As factors of heat, moisture and available food supply change from season to season, it is expected that mycological floras, both saprophytic and parasitic, may change. With a view, therefore, of learning whether the same technique would reveal different percentages of infection under varying seasonal conditions of succeeding summers, it was determined to make another survey during the same period of 1910. The same plan of germinating sections of wheat culms sterilized exteriorly as in 1909 was again followed. It was found that a great difference was to be noted. Following are the results for both internodes and nodes:

I. INTERNODES INFECTED. 1910. STERILE

| Plot Number | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | 14 | 15 | 16 | 17 | 18 | 19 | 20 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Colletotrichum sp. | 20 | 30 | 0 | 10 | 20 | 0 | 0 | 10 | 20 | 10 | 0 | 20 | 10 | 10 | 0 | 0 | 0 | 10 | 0 | 0 |
| Fusarium sp. | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 10 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Macrosporium sp. | 30 | 60 | 70 | 60 | 60 | 40 | 60 | 50 | 50 | 20 | 10 | 50 | 0 | 10 | 0 | 20 | 50 | 20 | 40 | 20 |
| Helminthosporium sp. | 0 | 10 | 20 | 10 | 30 | 10 | 10 | 10 | 50 | 70 | 20 | 10 | 20 | 10 | 0 | 10 | 30 | 20 | 50 | 10 |

II. NODES INFECTED. 1910. STERILE

| Plot Number | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | 14 | 15 | 16 | 17 | 18 | 19 | 20 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Colletotrichum sp. | 10 | 10 | 0 | 20 | 20 | 0 | 0 | 0 | 20 | 10 | 20 | 0 | 30 | 40 | 0 | 0 | 0 | 20 | 0 | 0 |
| Fusarium sp. | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Macrosporium sp. | 80 | 70 | 60 | 50 | 20 | 30 | 50 | 30 | 30 | 30 | 20 | 70 | 0 | 10 | 0 | 20 | 80 | 10 | 50 | 30 |
| Helminthosporium sp. | 0 | 10 | 30 | 30 | 50 | 0 | 30 | 0 | 50 | 60 | 20 | 30 | 30 | 20 | 10 | 10 | 40 | 20 | 40 | 10 |

In order to show at a glance a comparison of these results of varying seasons the following is appended :

| | 1909 | 1910 |
|---|---|---|
| Internodes | | |
| Colletotrichum sp. | 52.5 | 8.5 |
| Fusarium sp. | 27.5 | 0.5 |
| Macrosporium sp. | 53.0 | 36.0 |
| Helminthosporium sp. | 34.5 | 20.0 |
| Nodes | | |
| Colletotrichum sp. | 57.0 | 10.0 |
| Fusarium sp. | 33.5 | 0.0 |
| Macrosporium sp. | 53.5 | 37.0 |
| Helminthosporium sp. | 40.5 | 24.5 |

It will be noted that the results of 1910 are much lower than those of 1909. Also the season was much more of the semi-arid type. It is quite likely that the harder, drier straw of the dry season was less favorable for parasitic fungous growth than that of the more moist season. Physiological factors, both as regards host and parasite, are then to be considered. This is especially notable in the case of Fusarium and Colletotrichum.

Following is a very brief meteorological summary of the months of the growing period for the years 1909 and 1910.

These results were furnished by the courtesy of Mr. H. W. Grasse in charge of the Moorhead, Minn. Station of the U. S. Weather Bureau. This station is approximately one and one-half miles from the Agricultural College Experiment Farm.

| 1909 | May | June | July |
|---|---|---|---|
| Precipitation | 5.02 | 3.42 | 2.92 |
| Clear days | 15 | 15 | 17 |
| Partly cloudy days | 12 | 11 | 13 |
| Cloudy days | 4 | 4 | 1 |
| Mean temperature | 54 | 65.2 | 69 |
| **1910** | | | |
| Precipitation | 0.92 | 0.83 | 0.83 |
| Clear days | 20 | 20 | 24 |
| Partly cloudy days | 5 | 7 | 5 |
| Cloudy days | 6 | 3 | 2 |
| Mean temperature | 51.4 | 69.1 | 71.4 |

Very briefly, then, from the above it may be observed as follows:

| | 1909 | 1910 |
|---|---|---|
| Precipitation | 11.36 | 2.58 |
| Clear days* | 15.7 | 21.3 |
| Partly cloudy days* | 12 | 5.7 |
| Cloudy days* | 3 | 3 7 |
| Mean temperature* | 62.7 | 64 |

It will be noted from the above that growing conditions for host, and consequently parasite, were very different, there being approximately four and one-half times the rainfall during the season of greater infection than during the period when less infection was present. The amount of light, which is known to be a negative factor as regards mycelial growth, was much greater in 1910 than 1909. Likewise, in the mean temperature varied slightly.

The seed used each year on the plots had the same pedigree, the seed from each plot being sowed again on that plot the next year after cleaning. The wheat was mostly Fife. The factor of changing source of seed is not to be considered therefore. Possible change in food supply could not be determined.

For the purpose in view and because of paucity of statistics nothing can be said of the relationship of the soil treatment of these various plots with fertilizers, fungicides and germicides to the occurrence of these fungi on the wheat.

It is very likely that a worthy question for further study would be found in similar work regarding other parts of the plant. The seeds doubtless would show a different relationship. The more distal parts of the plant would have the greater likelihood of air infection since the rise of parasitic mycelium from soil infection as the plant matures would tend to be checked by the hardening and drying of the tissues. Especially would this be the case when very rapid growth of the plant had preceded a period of drought and consequent hardening of tissues previously developed. Time was lacking, however, for the study of this specific point.

Finally, we took the roots from some of the plants, selecting those especially which seemed to show some loosening of the cortex as though some disease condition had obtained. Such specimens of wheat root systems were sterilized thoroughly to do away with all surface infection, and then allowed to germinate in damp chambers. In nearly all cases, Colletotrichum, Fusarium or Macrosporium developed, showing root infection. As in the nodes and internodes the infection was usually a mixed one.

* Average for three months.

Check tests from virgin soil did not show these fungi in any large amount. Additional evidence is thereby adduced that part at least of the deterioration in yield in wheat fields in the Northwest may be due to infection from soil fungi or the spores of the same in wind-blown diseases. Confirmatory results have been obtained from numerous samples of wheat straw procured in widely scattered areas of North Dakota.

This survey seems then to show that:

First,—Old wheat soil is infected with certain fungi which are not present in large numbers in virgin soil.

Second,—The spores or mycelium of certain of these fungi are to be found normally in or on the wheat plant grown on such land.

Third,—A certain percentage of the wheat is pathologically infected with certain of these fungi.

Fourth,—It is likely that changes in meteorological conditions such as light, heat and moisture produce marked differences in total infection.

Fifth,—Certain of these fungi cause root infections.

AGRICULTURAL COLLEGE,
NORTH DAKOTA

# PRELIMINARY NOTE ON "SILVER LEAF" DISEASE OF FRUIT TREES

H. T. Güssow

(With Plate XXV)

In November, 1909, I observed in Nova Scotia the first authentic case, in this country, of a disease to which, in England, the name of "Silver Leaf" is given. In this case the disease occurred on an apple tree growing in the orchards of the Truro Agricultural College. I may say that Prof. Smith, the biologist of this college, pointed the tree out to me, wishing to ascertain whether I considered its condition identical with the European "Silver Leaf" disease. I have since observed at least sixty different trees in various localities similarly affected. From Nova Scotia to Vancouver Island the disease is known to me to exist, and it seems opportune to prepare a short note calling the attention of my United States colleagues to its widespread existence at least in Canada. Curiously enough I failed to discover any previous reference to its occurrence on this side of the Atlantic, nor was I able to ascertain its presence in the United States by frequent conversations on the subject. There are some references in the United States Experiment Station Record, but they are all taken from European literature. There must be some misconception somewhere, for it is hardly possible, that in Canada we should have so many cases, and none should be known of across the border. Let us hope that this is the case—but I have reason to doubt it. At any rate, special attention to this disease is very desirable.

"Silver Leaf" disease manifests itself by the turning silvery of the foliage of all kinds of fruit trees and shrubs. Plums, apples, pears, peaches, cherries, currants, gooseberries and others have been known to be attacked, but as yet in Canada, plums and apples only seem affected. The glaucous, often silvery, gloss of some of our native poplars and willows is well known to all. The leaves of trees affected with "Silver Leaf" show a similar milky or silvery gloss, which is totally distinct by its persistent nature from any similar colour of leaves due to hairiness or various spraying fluids. Some trees are so silvery in appearance that their abnormal condition is apparent from a great distance. On closer examination of the leaves of affected trees, they will be found to be covered with a rather brittle upper epidermis, but otherwise they are quite normal in size and shape. Microscopical surface examination reveals slightly coarser cell walls than are present in healthy specimens and sections through the leaves show a raising of the

epidermis above the underlying cells, resulting in an air space being formed between the chlorophyl-bearing and the cuticular cells. This air space is apparently responsible for the silvery gloss of the leaves. No organism has been observed in affected leaves by myself and others who have studied this disease. The silvery gloss may be apparent from the moment of the unfolding of the leaves, but it may be observed that the sound leaves may gradually assume this colour. At first not more than one or two limbs are affected, but in due course the leaves of the whole tree become silvery and slowly the tree succumbs. I may at once say that this is the general course of the disease, though in my experiments I have seen an infected branch recover. The colour though very distinct to one acquainted with this disease sometimes is not so pronounced and hardly recognizable. But experience soon teaches one to recognize the presence of the disease, even without any microscopical examination. There may be some indications of root trouble, stem or branch wounds or other mechanical injuries noticeable, but often no other sign is apparent until the tree or some of its branches are quite dead.

## RECORDS OF THE DISEASE IN OTHER COUNTRIES

*Germany*—Sorauer to whom this disease is well known gave to it the name "Milchglanz" (milky gloss). He speaks of it on page 285 of Vol. I, of his Handbook of Plant Diseases, Third Edition, 1909, to this effect :

The disease manifests itself on fruit trees ; the leaves though otherwise normally developed, lose their dark green appearance and show a silvery whitish affection. As a rule, a few limbs only suffer, beginning in June or July. In the second or more rarely the third year following the appearance of "Silver Leaf," the limb dies. In specimen trees which were examined again a year after removing any dead limb, the disease showed itself repeatedly in other branches. Thus he forms the provisional conclusion : "Silver Leaf" is an absolutely reliable symptom predicting the death of a limb.

The late Dr. Rud. Aderhold in Vol. V, Zeitschrift für Pflanzenkrankheiten, page 86, deals with the same disease at greater length. Both of these authors express the opinion that "Milchglanz" is due to a disturbance of the vegetative metabolism.

*France*—In France Dr. Edouard Griffon and Prof. Prillieux kindly inform me that the disease is generally designated by the name "plomb." It is known in various fruit trees, but has not been made the subject of any important study. The late Dr. Delacroix in his Vol. I (Maladies non parasitaire) of Diseases of Cultivated Plants, refers to it by the same name on page 227.

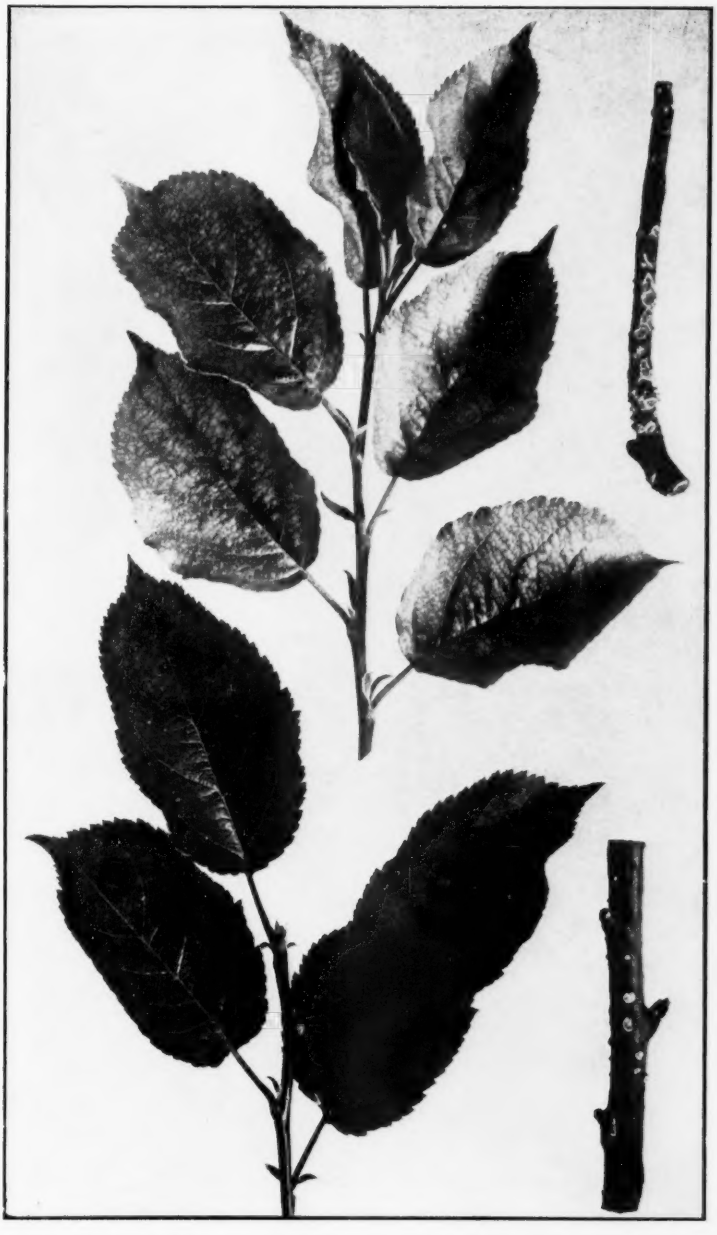


PLATE XXV—**Silver Leaf Disease of Fruit Trees**

The upper twig shows the characteristic silvery appearance of leaves affected by Silver Leaf, as compared with the dark green foliage from a sound tree below. To the right two small pieces of two-year-old apple wood, with the fungus *Stereum purpureum*, which appeared after the death of the branch from Silver Leaf. Photograph H. T. G.

*England*—In England this disease is known as "Silver Leaf" and it has there received considerable attention. Massee confesses he knows absolutely nothing of "Silver Leaf" but he states that he can produce it--not by infection—but by planting trees close to a drain from a stable. Percival has been the first to point out the probable cause of the mystery which he attributes to the fungus *Stereum purpureum*.

Pickering of the Woburn Fruit Experimental Farm repeated Percival's inoculation experiments with *Stereum* and again shows that this fungus is closely associated with "Silver Leaf." In England I observed an interesting case of true "Silver Leaf" in *Laburnum vulgare* growing in the garden of Dr. Carruthers, F.R S. After keeping the tree under observation for some years it finally had to be taken out because of its dying condition, when *Stereum purpureum* broke out all over the old stem. The inoculation of sound trees with portions of the hymenial layer of this fungus invariably produced "Silver Leaf" in all these trees. But it is not my intention to go into this question with too much detail at present. From South Africa come reports of "Silver Leaf," *Stereum* being found in many cases. From New Zealand I have the same information. In November, 1909, I noticed the first "Silver Leaf" tree in Canada, and asked Prof. Smith to look out for the fructification of *Stereum*, which he actually sent me a few months later stating that it appeared all over the part of the branches, now dead, that previously showed "Silver Leaf."

We have at present some thirty or more "Silver Leaf" trees in our orchards, plums and apples, in all stages—some plums with completely silvery foliage, even the young suckers growing from the roots of the parent plant. I have again collected *Stereum purpureum* from these branches. Inoculations were made last November with fresh *Stereum* tissue, bearing many ripe spores, and every tree thus inoculated showed plain signs of "Silver Leaf" as soon as the foliage appeared this spring. Check inoculations with a *Bjerkandera* and *Schizophyllum* very common also on dead and dying limbs and stumps did not result in the production of "Silver Leaf." The disease is under careful observation, but I have nothing as yet to say of its cure. Spraying appears on the face of it impracticable—the seat of the injury in many cases is the root—hence all our efforts should be concentrated upon prevention. The disease as Sorauer correctly states is a reliable sign predicting the death of the attacked trees which opinion other and my own experiments fully confirm. Branches and trees showing "Silver Leaf" should be removed and burned without delay.

OTTAWA, CANADA

# INJURY TO PINUS STROBUS CAUSED BY CENANGIUM ABIETIS[1]

BRUCE FINK

(WITH PLATE XXVI)

Early in the summer of 1909 a group of six white pine trees (*Pinus strobus* L.) on the grounds of The Western College for Women, at Oxford, Ohio, was found to be in a diseased condition. On the nineteenth of June, examination showed a large proportion of the leaves and twigs on the northeast sides of three of the trees to be dead or dying. The affected areas extended over the lower half or two-thirds of each tree, while the opposite side and the upper portion of the crown were of normal appearance. Blackish, roughly spherical tubercles from 0.5 to 1.25 mm. in diameter were found on the twigs bearing the dead leaves. The areas bearing the tubercles were brown and often shrunken. The tubercles were usually erumpent and subsessile, but some of them were just breaking through the periderm. With the tubercles, were found saucer-shaped bodies, of light color within the cavity, and it was found that most of the tubercles opened into the saucer-shaped form when moistened.

On some of the affected twigs, only a few leaves just back of the terminal bud were dead and of the characteristic red color. The tubercles were not present on these. In other instances all of the leaves of a twig were dead and the tubercles present. The terminal bud was dead on all of the affected twigs, and this seemed to indicate that the fungus was a parasite which gained entrance through or near the terminal bud. Microscopic examination showed the tubercles to be the fruits of the fungus commonly known as *Cenangium abietis* (Pers.) Dub. Besides the ascospores characteristic of this fungus, other long curved four-celled spores were seen occasionally, but the bodies in which they were produced were not found.

The diseased trees were from 1.5 to 2 feet in diameter four feet from the ground, and about 55 years old. They were situated at the crest of the hill about eight rods west of Peabody Hall. Fourteen white pines within a few rods of the diseased ones, but on lower ground, showed no trace of the disease, nor was the writer able to find evidence of it on any of the scattered conifers of the surrounding country, or in Oxford. Mr. J. T. Armistead, superintendent of grounds at The Western, afterward reported a group of four white pine trees near Oxford which died of the same disease. The two northeastward trees of the diseased group on The Western grounds were most affected and died in August of the year in which the disease was noted. The two

[1] Contribution No. 6 from the Botanical Laboratory of Miami University.

that died were on the highest ground and were probably injured more than the other trees by the severe drought of the previous year. Also the infection seems to have come from the northeast, in which case these two trees would be more exposed to it. They showed a more diseased condition than the other trees when first examined, though one of the trees shown in plate XXVI was badly infected. Mr. Armistead states that he had not seen evidence of this disease in previous years, but that he saw a few affected twigs on one of the surviving trees in 1910. The writer kept careful watch in 1910 to get spores for infection experiments, but failed to find the fungus.

All of the diseased twigs were removed and burned in June, 1909, and later in the same year, all the branches on the affected, northeast sides of the trees as high up as the disease extended, were cut off and burned. The accompanying plate shows the height to which the branches were removed from two of the surviving trees. In all but the most severely affected portions of the trees, the twigs with red leaves were interspersed with others bearing leaves of normal color. In July and August, the remaining leaves of the two trees that died turned brown. Other conifers were dying in 1909 as a result of the severe and prolonged drought of 1908, but none of these that the writer observed showed the peculiar mingling of red and green leaves characteristic of this disease, nor were the fruits of the fungus found on the other trees.

The 73 acre campus of The Western College has a deep soil, and, though well supplied with conifers and other trees, these two trees were the only ones that died during the years 1908 and 1909, at least on the several acres of planted woodland. However, it is not supposed that the death of the two trees was wholly caused by the fungus. Probably these pines at the hill-top were in an impoverished and non-resistant condition because of the drought of 1908, and the injury due to the fungus together with the effect of the drought killed the trees. It is of course impossible to know whether drought or fungus played the larger part in causing their death.

So far as can be ascertained this is the first reported instance of this fungus playing a part in killing trees in North America, and the first record anywhere of trees suffering severely from it, except in large areas of forest. From data received from Dr. W. G. Farlow and Dr. C. L. Shear, it appears that this fungus is not well known in North America, being represented in herbaria as follows: on *Pinus palustris*, Tuskegee, Alabama (G. W. Carver); on *Pinus sp.*, Nevada (C. F. Baker); on *Pinus ponderosa*, Colorado (F. E. Clements); on *Pinus insignis*, California (Harkness); on cones of *Abies excelsa*, Maine

(P. L. Ricker). Though the specimens are few, they indicate a wide distribution. Yet no injury seems to have been reported previously from America.

In 1887, the Austrian pine (*Pinus austriaca* Höss), which had been used for reforestation in western Norway was found to be largely dead or dying. Brunchorst[1] investigated and found it to be due to a fungus, which was named *Brunchorstia destruens* Eriks. Brunchorst gives a very good description of the fungus and its method of attack, and found that few if any of the branches escaped entirely. He thought that the circular areas about the bases of the leaf-pairs were the portals of entry. He also concluded that the fungus was the pycnidial stage of an Ascomycete, but he was not able to find ascospores. His paper is interesting and valuable, but more can not be given regarding it here.

According to Schwarz[2] *Pinus sylvestris* L. was badly injured by *Cenangium abietis* in eastern Germany in 1891 and 1892. Indeed, a considerable proportion of the trees were killed outright, and the losses were great. Schwarz made by far the most complete and valuable study that has appeared and treats the matter exhaustively. It is impossible, of course, to give anything of his excellent treatment, which gives both internal and external symptoms and the character of the fungus. Karsten had noted the same condition of pine trees as early as 1864, and Hartig had studied the matter later. The papers of Karsten and Hartig contain scarcely anything of value and are not cited herein. Schwarz differed from Brunchorst regarding the point of infection, believing it to take place through the terminal bud. The diseases in Norway and Germany are now known to have been due to different conditions of the same fungus.

The infection spreads from the point of entry in or near the terminal bud backward beyond the leaves of the twig into larger areas of the branch. In ours we found the oldest branches killed to be 15 years old and 1.5 cm. in diameter. It was found in Europe that the resin hardens in the branch back of the growing mycelium and usually stops the spreading of the latter backward into the larger branches and the trunks of the trees. Just what might have happened to our trees, had the branches not been cut off, is of course not known, but it is possible that the disease might have spread and appeared the following year in the four not killed, as such spreading sometimes occurs in Europe, according to Schwarz. On the other hand, it might have been sufficient to have cut off the affected twigs.

[1] Brunchorst, J. Ueber eine neue, verheerende Krankheit der Schwarzföhre (Pinus austriaca Höss) Berg. Mus. Aars., 1887. 6: 1-15. pl. 1-2. 1888.

[2] Schwarz, Frank. Die Erkrankung der Kiefern durch Cenangium abietis. 1-126. pl. 1-2. Jena, Gustav Fischer, 1895.

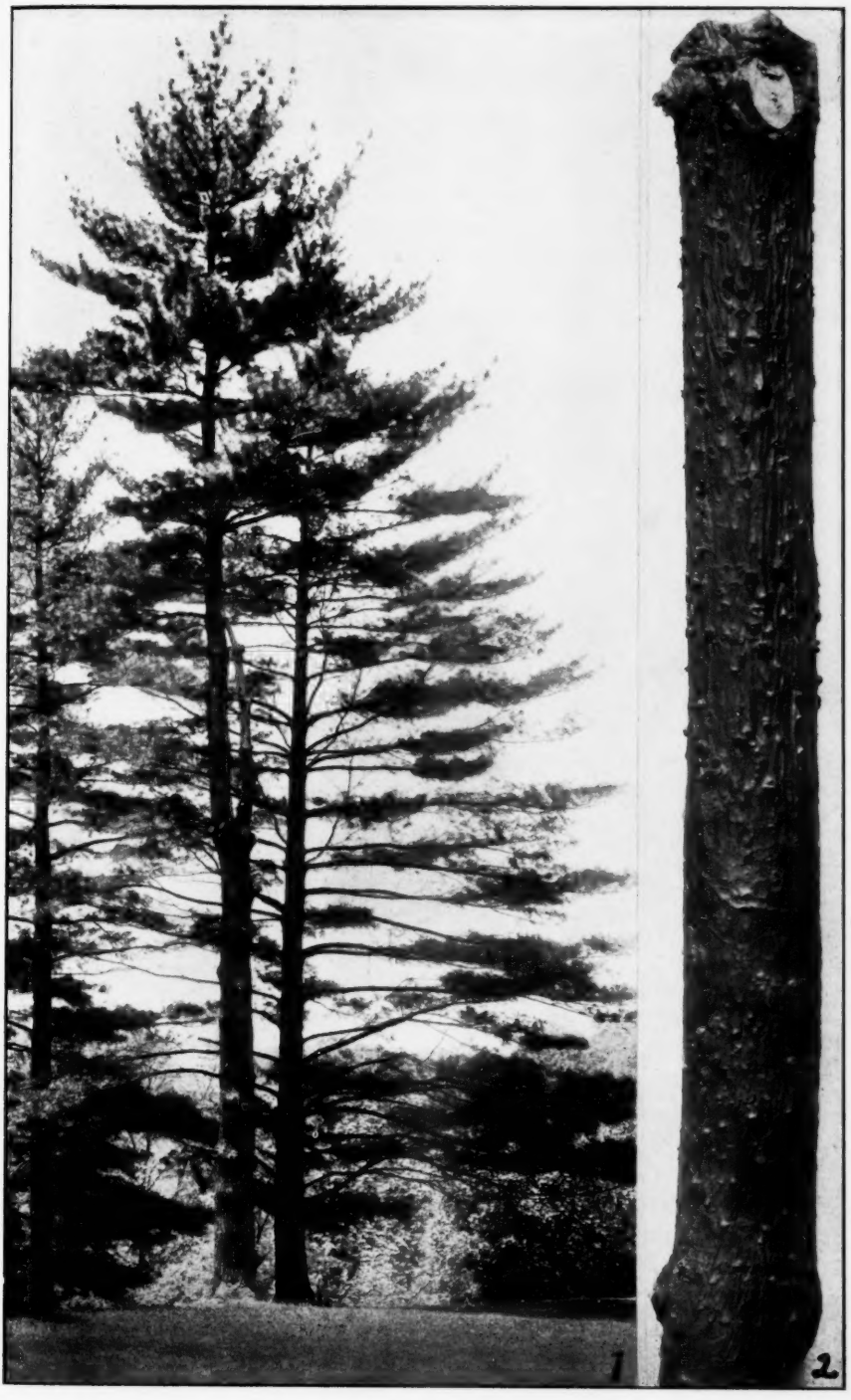


PLATE XXVI. **Cenangium Canker of Pine**

FIG. 1. Two of the trees still living, photographed after the infected limbs had been removed.

FIG. 2. One of the affected twigs showing the appearance of the apothecia and the shrinkage due to the destructive influence of the fungus. Multiplied two diameters.

As stated above we found the long 4-celled spores which correspond to those of the pycnidial stage noted by Brunchorst and described by Eriksson, but the bodies in which they are formed could not be found. The condition of our fungus was like that most commonly seen by Schwarz in that it showed the ascospore condition well developed. The apothecia are shown, partly opened and saucer shaped and partly closed, in plate XXVI. Schwarz found a third kind of spores, which are simple and hyaline. He found the fungus on several species of pine, on some of which the ascospores were not present, while on others they were the only kind found. Among the latter was *Pinus strobus*, and this corresponds well with our finding.

We were delayed in culture experiments so long after our plants were collected that the spores failed to germinate, and publication was postponed in the hope of securing fresh spores for infection experiments. Not succeeding in obtaining fresh specimens, the infection experiments have been abandoned for the present at least. A short account of the local infection was published[1] in The Ohio Forester, thinking that some observers might notice the disease, especially in one or more of the several areas in Ohio where pines abound. Though no response has resulted from the inquiry, there can scarcely be a doubt that this fungus has frequently caused damage to pines in various parts of North America, and that the diseased condition has been overlooked or ascribed to some other cause.

It is hoped that this paper may prove valuable in calling attention to a fungus which is now known to cause serious damage in planted groups of pines as well as in forests. The disease seems to occur only occasionally, after seasons of prolonged drought. Whenever found, the papers of Brunchorst and Schwarz cited herein will be found very valuable for the suggestions contained.

Rehm gives the following synonymy in Rabenhorst, L. Kryptogamen Flora $1^3$: Ascomyceten. 227-228 and 1255. *Peziza abietis* Pers. Syn. Fung. 671. 1801. *Cenangium ferruginosum* Fr. Vet. Akad. Handl. 361. 1818. *Peziza cervina* Pers. Syn. Fung. 647. 1801. *Tryblidium pineum* Pers. Myc. Eur. 332. 1822. *Cenangium abietis* Duby Bot. Gall. **2**: 736. 1830.

We are not concerned here with the matter of synonymy, and prefer to use the well known name of the fungus. However, according to the recent rules, *Cenangium ferruginosum* Fr. (Syst. Myc. **2**: 187. 1822) is the valid name.

[1]Fink, Bruce. A rare and dangerous Disease of Pine Trees. The Ohio Forester. 2: 5. 1911.

MIAMI UNIVERSITY,
OXFORD, OHIO

# TRICHODERMA KÖNINGI THE CAUSE OF A DISEASE OF SWEET POTATOES

MEL. T. COOK AND J. J. TAUBENHAUS

(WITH PLATES XXVII AND XXVIII)

The great number of rots to which the sweet potatoes in storage are subject makes them of special interest from both the scientific and economic standpoints. Among the most important of these pests is the so called "ring rot" which is especially common on the Delaware-Chesapeake peninsula. It occurs as a dry rot which spreads in all directions from the point of origin and completely girdles the root, thus forming a well defined ring or band of discolored tissue.

This disease was reported to the Delaware Experiment Station so often that in the fall of 1910 the writers began a study of the same to determine its cause and a remedy. Careful examination of a number of roots failed to reveal any sign of spores or other structures by which the organism could be determined. A number of diseased roots were carefully washed and the surface disinfected with formalin, cut with a sterilized knife and fragments of affected tissue transferred to poured plates of medium No. II [1] some of which were acidified and others not acidified. After four days pure growths of the same character appeared in all the plates. Within seven days the fungus had completely overrun the entire surface of the agar and had very much the appearance of a Fusarium (Fig. 13). Microscopical examinations proved the growth in all cases to be sterile. This fungus was transferred to slant tubes of medium No. II for preservation and further study. At the time the above culture was started a number of diseased potatoes were put in moist chambers. These roots underwent a very rapid decay and also produced dense surface growths of white mycelium of the same appearence as that produced on the plate cultures (Figs. 6, 7). When bits of this mycelium or bits of the decayed roots were transplanted to poured plates of medium No. II, the same growths were obtained as in the preceding cultures. After four or five weeks the mycelium on the potatoes gave a scanty spore formation which was somewhat more abundant on that part which had spread from the potato to the filter paper which was used to maintain the moisture. Spores were seldom obtained from the growth produced on medium No. II, but when grown on medium No. V spores were produced in abundance. The above experiments were repeated a number of times but always with the same results. The organism was at first supposed to be *Trichoderma lignorum* (Tode) Harz, but specimens of both cultures and diseased potatoes sent to Dr. Thaxter and Dr. Peck

[1] The composition of this and the other media used in these studies is given at the end of this article.

were determined as *Trichoderma köningi* Oudemans. The pathogenicity of the organism was determined as follows: Apparently healthy sweet potatoes were selected, carefully washed and disinfected by allowing to remain for 10 minutes in 5% formalin and then rinsed in sterilized water. These were put in sterilized moist chambers and kept for one week after which all that showed indication of this or any other disease[1] were discarded and the healthy ones inoculated from pure cultures of the organism. In these inoculations, small bits of the fungus were inserted into slits made by means of a sterilized scalpel. The checks were cut in the same manner and both inoculated roots and checks kept under the same conditions of moisture and temperature. These experiments were also repeated a number of times and in every case there was 100% infection within two weeks while the checks remained completely healthy. (Figs. 1, 2, 3, 4, 5). The diseased roots were eventually destroyed by the fungus. The above experiments were repeated in the same manner except that the potatoes were not injured, the organism being placed on the uninjured surface. In all cases the fungus grew over the surface forming a very fine mycelial growth but without penetrating the epidermis until it reached the cut end or other wound through which it was able to gain entrance. Similar results were obtained when diseased potatoes were placed in moist chambers with healthy potatoes, the fungus gradually spreading over the surface of the healthy roots until it reached a wound which afforded entrance. These last experiments were repeated using chambers without moisture but always with the same results. White or Irish potatoes were also inoculated with *Trichoderma köningi* but no infection took place. A number of sweet potatoes were also inoculated with *Trichoderma lignorum* which was obtained from Dr. Thaxter and gave 50% infections but with symptoms slightly different. (Fig. 8).

The symptoms of the disease may be given as follows: In their earliest stages the spots are circular and of a light brown color with a tendency to wrinkle (Fig. 1). The flesh is hard and water soaked, brown in color with a black zone at point of contact between the healthy and diseased tissue. The spot enlarges in all directions and eventually destroys the entire root. When the decay is well advanced, a very luxuriant white mycelial growth is formed on the surface. Spores are produced very sparingly on this growth when in contact with the decayed tissue but very abundantly on that part of the mycelium which has spread over the healthy surface or onto the glass or filter paper of the moist chamber. It will be readily seen that these

[1] The most common disease which appeared on these potatoes was the soft rot, due to *Rhizopus nigricans*.

symptoms are not exactly the same as the "ring rot" when found in the store houses. Whether this variation is due to another organism or to the use of pure cultures is a question which remains to be solved. The symptoms produced by *T. lignorum* were very similar to those of *T. köningi* except that a deeper brown zone separates the diseased from the healthy tissues (Fig. 8).

*Trichoderma lignorum* is common and widely distributed on decaying wood and various other substances. *Trichoderma köningi* was originally isolated from the soil by Oudemans[1], and is still looked upon as a soil organism. The spores of *T. lignorum* are spherical while the spores of *T. köningi* are elliptical (Figs. 16, 17). In germinating the spores of *T. köningi* send out from one to two germ tubes while the spores from *T. lignorum* in germinating send out two or three germ tubes. (Figs. 19–26). The mycelium of *T. köningi* is distinguished by its great abundance of chlamydospores (Figs. 39–42) while the *T. lignorum* very rarely produces chlamydospores (Figs. 43, 44). In very old cultures of *T. köningi* the chlamydospores are frequently free and in a state of germination (Figs. 31–33). What appear to be haustoria were seen in tissue affected with *T. köningi* (Fig. 46). The conidophores of the two organism did not show distinguishing characters (Figs. 47–53). When grown on different synthetic media the two organisms showed the following characters (Figs. 9–15).

COMPARISON OF TWO ORGANISMS ON SYNTHETIC MEDIA

| | *Trichoderma lignorum* | | | | *Trichoderma köningi* | | | |
|---|---|---|---|---|---|---|---|---|
| Medium | Zonation | Mycelium | Spores | Chlamydospores | Zonation | Mycelium | Spores | Chlamydospores |
| II | perfect | abundant | deep green | occasional | none | abundant | rare green | abundant |
| IV | " | " | " | none | " | fair | abundant deep green | none |
| VI | broad | " | " | " | " | " | irregular scattered green | abundant |
| XIV | narrow | " | " | " | " | scanty | few and scattered green | few |
| XV | perfect but later imperfect | " | " | " | " | abundant | rare green | abundant |

[1] Oudemans, C. A. J. A. Podrome d'une flore mycologique obtenue par la culture sur gélatine préparée de la terre humeuse du "Spanderswood," pèrs du Bussum. Archiv. Néerlandaises Sci. Exactes et Naturelles, series II, 1902 p. 291, plate XXXI figs. 1–7.

In all cases when grown on synthetic media the spore formation was most abundant around the margin of the glass.

## COMPOSITION OF MEDIA

### MEDIUM II

| | |
|---|---|
| Water | 1000 cc. |
| Agar | 15 grams |
| Glucose | 20 grams |
| Dipotassium phosphate | 0.25 gram |
| Magnesium sulphate | 0.25 gram |

### MEDIUM IV

| | |
|---|---|
| Water | 1000 cc. |
| Agar | 15 grams |
| Glucose | 10 grams |
| Peptone | 2 grams |
| Ammonium nitrate | 1 gram |
| Potassium nitrate | 0. 5 gram |
| Dipotassium phosphate | 0.25 gram |
| Calcium chloride | 0.01 gram |

### MEDIUM VI

| | |
|---|---|
| Water | 1000 cc. |
| Agar | 15 grams |
| Glucose | 10 grams |
| Liebigs beef extract | 10 grams |
| Ammonium sulphate | 2 grams |
| Dipotassium phosphate | 1 gram |

### MEDIUM XIV. CORN MEAL AGAR

15 grams corn meal cooked in 500 cc. water for one hour at 60° C.
Strain through cloth.
15 grams agar melted in 500 cc. water.
Mix the two and filter through cotton.
(Note.—Modification of method used in Bureau of Plant Industry of the U. S. Dept. of Agriculture.)

### MEDIUM XV. LIMA BEAN AGAR

Grind 50 grams of lima beans in meat cutter and soak for one half hour in 500 cc. tepid water. Simmer for one half hour. Strain.
Melt 15 grams of agar in 500 cc. water. Mix.

## SUMMARY

1. *Trichoderma köningi* and *T. lignorum* are both the causes of storage rots of the sweet potato but the former is of greater importance.

2. *T. köningi* is closely associated with the so called "ring rot" but when used from pure culture does not produce the typical rot.

3. *T. lignorum* produces spores much more freely than *T. köningi*.

4. *T. köningi* produces spores rarely on medium No. II but abundantly on medium No. V.

5. *T. köningi* produces spores on the sweet potato only when the rot is far advanced while *T. lignorum* produces spores abundantly at a very early stage of decay.

6. When grown in synthetic media *T. lignorum* is characterized by the zonary growth which does not occur in *T. köningi*.

7. Chlamydospores are much more frequent in *T. köningi* than in *T. lignorum*.

The writers wish to thank Dr. Charles Thom, Prof. Roland Thaxter, and Prof. Charles Horton Peck for courtesies in determining specimens.

DELAWARE COLLEGE AGRICULTURAL
EXPERIMENT STATION,
NEWARK, DEL.

## EXPLANATION OF PLATES

### PLATE XXVII

FIG. 1. Surface view of spot caused by *T. köningi*.
FIG. 2. Early stage of artificial infection of sweet potato with *T. köningi*.
FIG. 3. Later stage of Fig. 2.
FIG. 4. Same as Fig. 2 but more advanced stage of decay.
FIG. 5. Cross section of same stage as Fig. 2.
FIG. 6. Surface view of diseased sweet potato in an advanced stage showing the diseased area covered with the mycelium of *T. köningi*.
FIG. 7. Logitudinal section of Fig. 6, showing rotted area.
FIG. 8. Sweet potato artificially inoculated with *T. lignorum*.
FIG. 9. *a*, *T. lignorum* grown on medium No. IV.
*b*, *T. köningi* " " " " "
FIG. 10. *a*, *T. lignorum* grown on medium No. XV.
*b*, *T. köningi* " " " " "
FIG. 11. *a*, *T. lignorum* grown on medium No. XIV.
*b*, *T. köningi* " " " " "
FIG. 12. Young culture of *T. lignorum* grown on medium No. II.
FIG. 13. Young culture of *T. köningi* grown on medium No. II.
FIG. 14. *a*, *T. lignorum* grown on medium No. II.
*b*, *T. köningi* " " " " "
FIG. 15. *a*, *T. lignorum* grown on medium No. VI.
*b*, *T. köningi* " " " " "

### PLATE XXVIII

FIG. 16. Spores of *T. köningi*.
FIG. 17. Spores of *T. lignorum*.
FIG. 18. Free chlamydospores of *T. köningi*.
FIGS. 19-26. Different stages in the germination of the spores of *T. köningi*.
FIGS. 27-30. Different stages in the germination of the spores of *T. lignorum*.

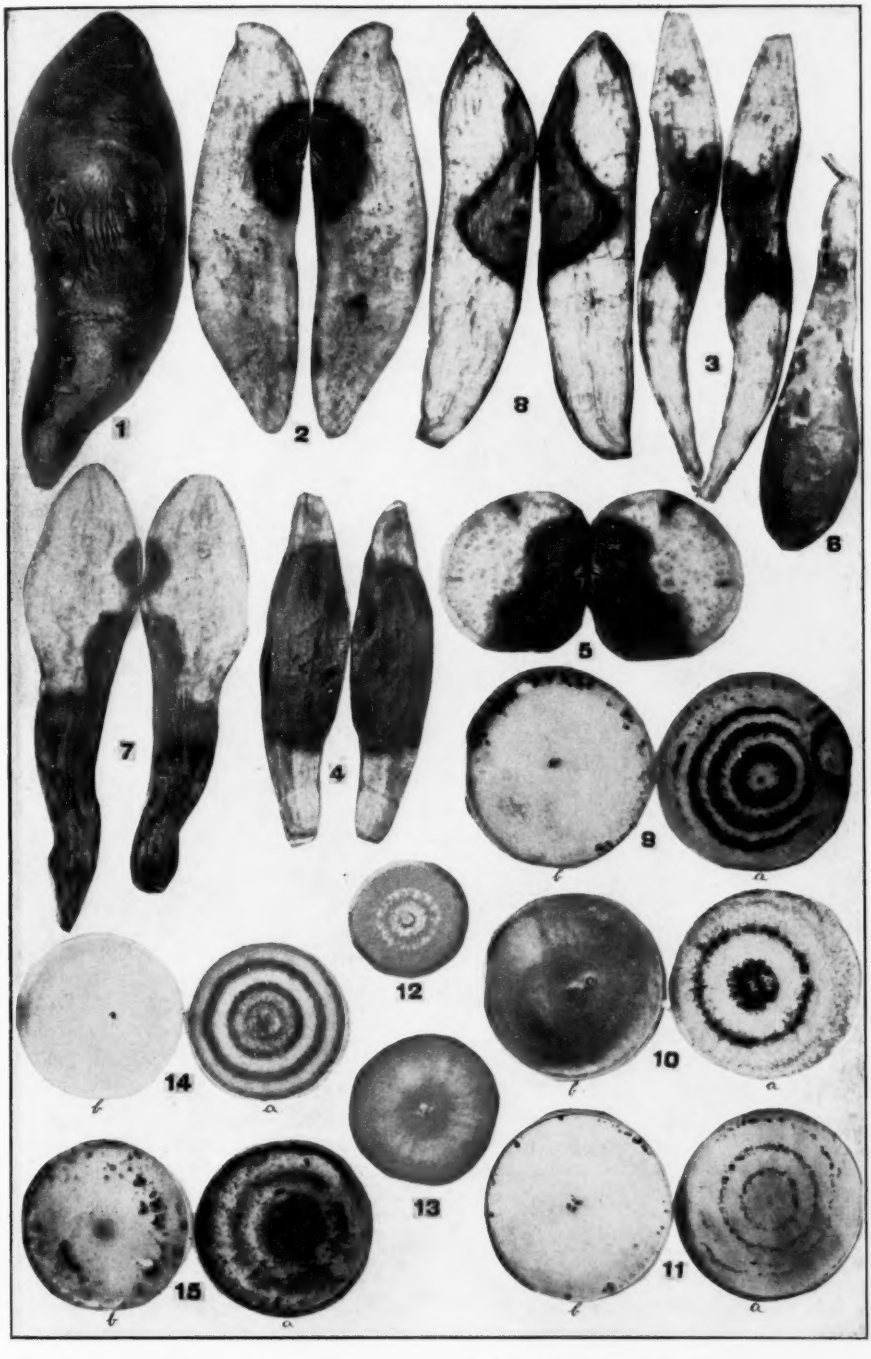



PLATE XXVII. **Trichoderma Köningi in Sweet Potatoes**

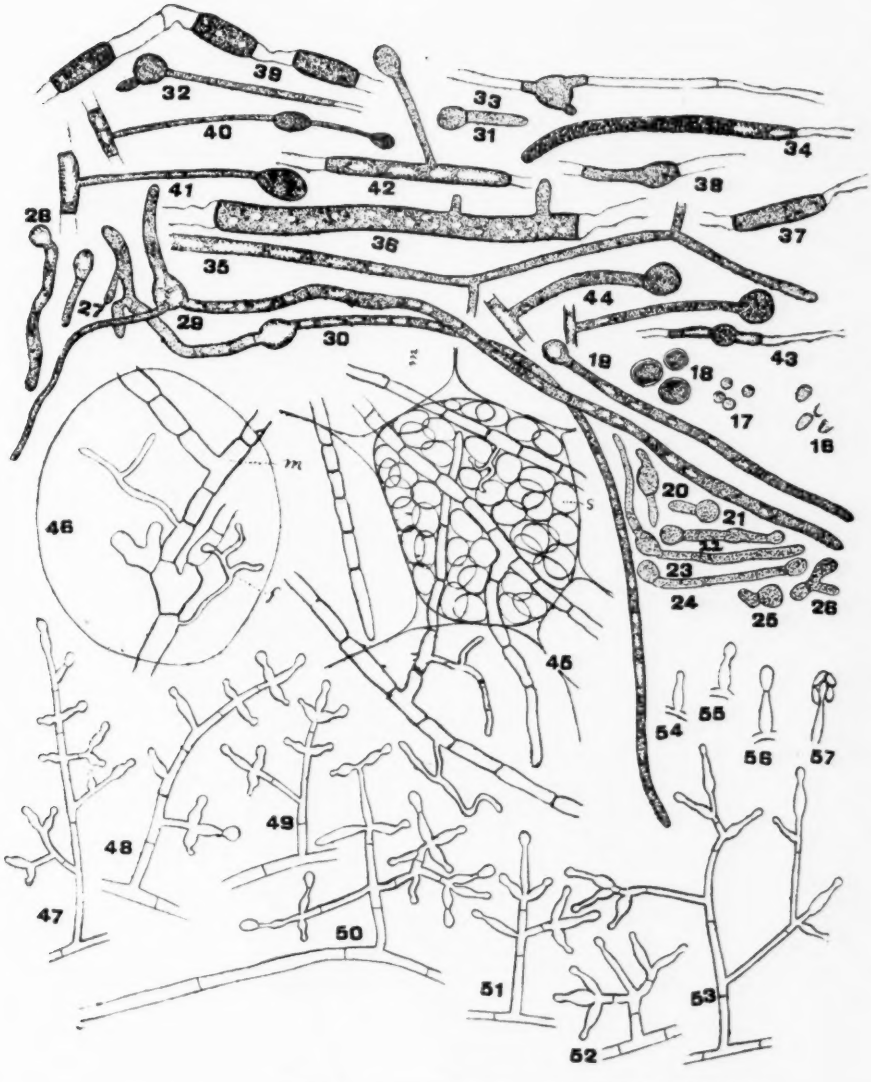



PLATE XXVIII. **Trichoderma Köningi in Sweet Potatoes**

FIG. 31. Germination of a free chlamydospore of *T. köningi.*

FIGS. 32–33. Germination of chlamydospores of *T. köningi* still attached to its mother mycelium.

FIG. 34. Young mycelium of *T. köningi.*

FIGS. 35–38. Old mycelium of *T. köningi* showing active cells of the hyphae full of protoplasm, while the neighboring ones are empty and beginning to shrivel.

FIGS. 39–42. Stages in chlamydospore formation in *T. köningi.*

FIGS. 43–44. Chlamydospores formation of *T. lignorum* on medium No. II.

FIG. 45. Cross section of infected tissue of sweet potato showing single cells invaded with mycelium of *T. köningi*; *s*, starch grains; *m*, mycelium.

FIG. 46. Single cell of infected sweet potato showing mycelium of *T. köningi*, with filaments resembling haustoria; *m*, mycelium; *f*, filaments resembling haustoria.

FIGS. 47–49. Conidiophores of *T. lignorum.*

FIGS. 50–53. Conidiophores of *T. köningi.*

FIGS. 54–57. Stages in the formation of the conidia of *T. köningi.*

# VARIATION OF VARIETIES OF BEANS IN THEIR SUSCEPTIBILITY TO ANTHRACNOSE

MORTIER F. BARRUS

(WITH PLATE XXIX)

Statements have often been made that varieties of beans vary in their susceptibility to the anthracnose; that wax-podded varieties are more susceptible than green-podded beans; that newer varieties are more nearly immune; and that the smoother skinned and more succulent varieties are oftener attacked than the coarser ones. The writer has noticed that some varieties of beans appear much more susceptible to anthracnose than others and after taking notes on the presence of this disease on the various varieties concluded that certain ones often appeared entirely immune simply because they had not been inoculated with the spores of the fungus. Evidently the only reliable method of determining the relative susceptibility of the various varieties to anthracnose is to inoculate them with the spores of the causal organism under conditions favorable to infection. This can best be accomplished by spraying the plants with a suspension of spores and covering them with a shaded bell-glass for 18 to 24 hours, or, under field conditions, by inoculating them at the beginning of a rainy period of a day or so, preferably in the evening. It is true, however, that under conditions apparently less favorable good infection often results.

During the spring of 1910 seed of as many varieties as possible (161 varieties) was obtained and these were planted in hills in the field at Oneida, N. Y., and a duplicate set in tin cans placed side by side in the soil out of doors. After making from four to seven inoculations on each of these varieties it could be seen plainly that there was apparently a great variation among the varieties in their susceptibility to this disease. Some varieties became very badly infected as a result of each inoculation, while others were not at all affected, and there were all gradations between the two extremes. Each successive inoculation produced similar results in most cases but some of the varieties were more affected after certain inoculations than after others. These cases, in the writer's opinion, should be explained as due to the inconstancy of environmental factors, and to different degrees of maturity of the plants rather than to a variation in the power of the fungus to infect these plants. Below, Table I, is shown some of the results of these inoculations in tabular form.

The data of the seventh inoculation of 1910 are missing but the results of two other inoculations made in 1911 in the greenhouse are in-

cluded. The inoculation of July 28 was followed by fair weather instead of damp cloudy weather as was expected, but the infection differs only in degree from the others. When inoculated under greenhouse conditions those varieties which had hitherto shown no infection, showed a slight infection in some cases. This may be due to the more favorable conditions for the fungus rather than to any unfavorable conditions for the host plant. Burger's Stringless is one of the few cases where there was no uniformity in the infection, but the fact that fair to excellent infection was obtained in four inoculations is sufficient evidence that the variety is a susceptible one.

The results of these experiments show that by far the larger number of varieties of *Phaseolus vulgaris* are more or less susceptible ranging from Detroit Wax, Imperial Wax, Fuller's White Wax, Blue Pod Butter, and Warren which showed little or no infection, to Lima Wax, Golden Refugee, Navy Pea, and several other varieties which became badly infected. There were about ten varieties which became but slightly infected even under favorable conditions. These were Red Marrow, Bountiful, China Red Eye, Red Kidney, White Kidney, Indian Chief, Child's Horticultural, Early Horticultural and Worcester Mammoth. A smaller percentage of the pole than of bush varieties of *P. vulgaris* were affected and the wax were scarcely more susceptible than the green-podded varieties.

An interesting instance of susceptibility and resistance to disease is shown in the case of a hill of Kentucky Wonder growing beside a hill of Early Horticultural, both climbing green-podded varieties. The vines of one variety wound about those of the other as they grew in the field. Both hills were inoculated at the same time and under similar conditions. The vines and pods of Kentucky Wonder were badly spotted with anthracnose contrasting sharply with the closely entwined healthy vines of the Early Horticultural.

Inoculation of other species of beans showed no infection on *Dolichos sesquipedalis* and *Vicia faba*, but excellent infection on the leaves of *Phaseolus multiflorus* var. Scarlet Runner. Of *Phaseolus lunatus macrocarpus*, slight infection was obtained on the leaves and pods of Burpee and of Wonder, and fair infection on leaves and pods of Burpee Improved and of Leviathan. A slight infection was obtained on the leaves of *Phaseolus lunatus* var. Henderson. For the most part varieties of these species were quite resistant to anthracnose and the infection obtained was not of sufficient consequence to be of commercial importance.

During the fall of 1910, graduate students in Plant Pathology, as part of their work in course, isolated *Colletotrichum lindemuthianum*

TABLE I.—*Showing general uniformity of results from eight separate series of inoculations with strain A, from pure cultures. Series 1–6 inclusive were made during 1910, Series 8 and 9 during 1911.*

| Series | 1 | 2 | 3 | 4 | 5 | 6 | 8 | 9 |
|---|---|---|---|---|---|---|---|---|
| VARIETY | *Can* Inoc. Jly. 21 Obs. Jly. 28 | *Field* Inoc. Jly. 25 Obs. Aug. 2 | *Field* Inoc. Jly. 28 Obs. Aug. 5 | *Field* Inoc. Aug. 5 Obs. Aug. 19 | *Field* Inoc. Aug. 10 Obs. Aug. 19 | *Can* Inoc. Aug. 31 Obs. Sept. 9 | *G. house* Inoc. Apr. 13 Obs. Apr. 21 | *G house* Inoc. Apr. 5 Obs. Apr. 13 |
| Burpee White Wax | Good | Excellent | Excellent | | | Excellent | | |
| Currie | Good | Excellent | Excellent | | | Good | | |
| Detroit | None | None | None | None | None | None | Slight | Very slight |
| Golden Eyed Wax | Excellent | Excellent | Fair | | | Good | | |
| Imperial | | None | None | Very slight | | None | Very slight | |
| Refugee Wax | Excellent | Excellent | Fair | | | Excellent | | Excellent |
| Wardwell | | Excellent | Fair | | | Good | Excellent | Excellent |
| Blue Pod Butter | None | None | None | None | None | None | None | Very slight |
| China Red Eye | | None | None | None | None | None | Very slight | Very slight |
| Golden Refugee | Excellent | Fair | Good | | | Good | | Excellent |
| Navy Pea | Excellent | Excellent | Fair | | | | Excellent | Excellent |
| Red Kidney | | None | | None | Slight one pod | None | None | Very slight |
| Turtle Soup | Excellent | Fair | Slight | Excellent | Excellent | Excellent | Excellent | Excellent |
| Warren | None | None | None | None | None | Slight | Slight | |
| Burger Stringless | Excellent | None | None | Exclt. in 1 plant | Fair | Excellent | | |

(Sacc. & Magn.) Bri. & Cav. from diseased beans obtained from the disease garden and with these cultures inoculated a few of the varieties of beans which had proved susceptible and also one of those that had hitherto proved to be resistant in the experiments. To the surprise of all the resistant variety became infected. Cultures were then obtained from these men and various varieties hitherto designated resistant and susceptible were inoculated with these new strains and also with a culture of the fungus which was used by the writer during 1910 and which was isolated from diseased beans obtained at Oneida, N. Y.

The results of those experiments are indeed interesting, for, almost without exception, the varieties which had proved to be resistant to this first strain of the organism gave excellent infection from inoculations with the other strains, and what is most surprising the varieties Navy Pea and Golden Refugee which had given excellent infection to the first strain (Table I) gave slight or no infection when inoculated with the second strain (Table II and compare Figs. 1 and 2 in Plate XIX). This indicates that the difference between the strains is not one of degree in virulence but is inherent and absolute and is possibly an adaptation to particular varieties as hosts.

These experiments were repeated several times and always with the same general results, making it safe to conclude that there is a real difference in the infecting power of the strains. Other cultures of the organism were obtained from various sources and inoculations upon different varieties of beans were made with each. The results of some of these are given in tabular form below (Table II).

It can be seen from the table that Culture $A_1$ and $A_2$ differ in the majority of cases from the other cultures in their ability to infect the various varieties. $A_2$ is a culture isolated from a plant inoculated with $A_1$. Cultures B, C and D produced practically the same results and probably represent one strain. It was found upon looking up the source of the cultures that B and C were obtained by different men from the same garden and the beans from which the cultures were obtained were probably infected by spores from the same source. Culture D was obtained from a Detroit bean seedling grown in the greenhouse at the college from seed produced at Oneida the summer before, this variety there showing no sign of anthracnose on vines or pods during the entire summer. It may be that the seedling had become infected accidentally from spores of culture B or C. Culture E was obtained in the spring from the surface of a diseased bean pod kept over winter in an attic. It affects the varieties similarly to cultures B, C and D but to a less degree and perhaps should be reported as a separate strain.

As is shown in Table I there is a great uniformity in the extent of infection produced on the different varieties from the inoculations made at different times, although the last inoculation was made more than ten months after the first, during which time the fungus was transferred to fresh media a number of times. This culture was isolated in the spring of 1909, but it has lost none of its virulence by being in culture so long. In order, if possible, to increase its virulence, susceptible varieties were inoculated and cultures made from lesions produced on the stems. Susceptible and resistant varieties were then inoculated with spores from this new culture, designed $A_2$, but the results were remarkably similar to those obtained with $A_1$ which had been in culture for two years. (Table II).

TABLE II. *Showing the comparative results of inoculations of a number of varieties of beans with different strains of C. lindemuthianum.*

| Varieties | $A_1$ | $A_2$ | B | C | D | E |
|---|---|---|---|---|---|---|
| Detroit | None to very slight | Very slight | Excellent | Excellent | Excellent | Fair to excellent |
| Imperial | None to very slight | | Excellent | Slight to good | Excellent | |
| Refugee Wax | Excellent | Excellent | Good | | | |
| Wardwell | Excellent | | Excellent | | Good | Fair to excellent |
| Blue Pod Butter | None to very slight | Very slight | Excellent | | Excellent | Slight to excellent |
| China Red Eye | None to very slight | Very slight | Excellent | Excellent | Excellent | Slight to fair |
| Navy Pea | Excellent | Excellent | Slight | Slight | None | None to slight in 3 cases |
| Red Kidney | None to very slight | Very slight | Excellent | Excellent | Good to excellent | Fair to excellent |
| Turtle Soup | Fair to excellent | Fair to excellent | Fair to good | | Fair to excellent | None to fair |
| Warren | None to very slight | | Excellent | Good to excellent | Fair to excellent | |
| White Kidney | None to slight except 1 case | | Excellent | Excellent | Excellent | |
| Yellow Cranberry | Fair to excellent | Fair to excellent | Excellent | Excellent | Excellent Slight in 2 cases | Good to excellent |
| Golden Cluster | Excellent | | Fair to good | | | None |
| Red Marrow | None to slight | | Excellent | Good to excellent | Excellent | Slight to fair |
| Golden Refugee | Excellent | Excellent | None | | | Very slight |

It must be admitted that in many instances not noted in the tables, the degree of the infection obtained by the various strains is not strik-

FIG. 1. Five varieties of beans inoculated with spores from culture A. Reading from left to right these are, Detroit, Imperial, Blue Pod Butter, Navy Pea, and China Red Eye.

FIG. 2. Same five varieties inoculated with spores from culture B. Same order as in Fig. 1.

PLATE XXIX. **Strains of C. lindemuthianum on Varieties of Beans**

ingly different, but this does not detract from the significance of the several cases where the difference is clearly marked and continues so. It does indicate that there is a distinct difference between cultures of anthracnose, obtained from diverse sources, in their power to infect the various varieties of beans, and, while the experiments are not yet extensive enough to warrant this conclusion, they seem to indicate that different strains of the fungus are regional in their distribution; that in certain regions one or several strains may be found which are distinctly different in their infecting power from strains obtained in other places. It is true that through the distribution of diseased seed by seedsmen, the various strains have been widely scattered. May we not, from these experiments, conclude, however, that it is not sufficient to say that because a variety has proved to be free from anthracnose in a few regions it is necessarily immune to this disease? A variety which in a given region has proved immune may when introduced into another locality just as favorable to the growth of beans and no more favorable to the fungus, become badly diseased, due to the presence there of another strain of the parasite.

It is possible that various strains of other fungi may differ in their power to infect the host plant and that certain plants now heralded as immune may be susceptible if attacked by a strain of the fungus from another source. This may explain why some plants ordinarily regarded as immune become susceptible when grown in certain regions, although the influence of environmental factors upon the host should not be disregarded.

It may be stated in conclusion, therefore, that the writer has not found any varieties of *Phaseolus vulgaris* that have been resistant to every strain of the bean anthracnose fungus tested. Some, it is true, are less susceptible than others and it may be that certain varieties that have not yet come under observation may prove entirely immune. The writer does not wish to appear so pessimistic as to intimate that it is very unlikely that any one shall be able to find a resistant strain of any variety. It is to be hoped that such a strain may be found but before we can be sure of its immunity it will be necessary to test its ability to withstand infection from the various strains of the organism. Simply because a plant appears to be free from disease when its neighbors of the same variety have succumbed is enough evidence to arouse suspicion but not enough to prove the case.

CORNELL UNIVERSITY,
ITHACA, N. Y.

# A STUDY OF SOME GLOEOSPORIUMS AND THEIR RELATION TO A SWEET PEA DISEASE[1]

J. J. TAUBENHAUS

(WITH PLATE XXX AND TEXT FIGURES.)

A very serious anthracnose disease of the sweet pea on the Delaware Experiment Station farm was called to the attention of the writer during the latter part of July, 1910. This disease proved to be the same or very similar to the one reported by Sheldon[2] from West Virginia in 1905, and is very probably the so called "wilt" which has been so often referred to in old seed catalogues and treatises on sweet peas.

*Symptoms.* The disease occurs on the stems, flowers and pods but is most severe on the latter. There is a general wilting of the affected parts followed by dying, which begins at the tips of the younger shoots and works downward. The older parts of the plants are not killed immediately but may persist for some time after being attacked by the fungus. The dead parts shrivel, become brittle, and are soon covered with minute acervuli. The affected pods are at first a dirty white in appearance but assume a dull color, which is due to the presence of the acervuli. A definite canker, which is so characteristic of the bean anthracnose is not produced. Although the disease on the stems seems to be restricted to the youngest growths, the pods may be infected at any stage of their development. The seeds of the diseased pods are always infected, become shrivelled and frequently do not reach maturity.

*Pathogenicity.* Sheldon called attention to the identity of the Gloeosporium of the sweet pea with *Glomerella rufomaculans* (Berk.) Spauld. & von Sch. which is the cause of the bitter rot of the apple. With this in mind, the writer made the following experiments in the autumn of 1910. Sweet pea seeds, which to all appearances were perfectly healthy, were carefully selected, sterilized by immersion for 15 minutes in a 5% solution of formalin, and planted in pots in soil, which had been sterilized by heating for one hour in the autoclave. The seeds germinated in 5 days and the seedlings were allowed to grow for three weeks. Fifty plants were allowed to grow in each pot. The temperature of the room ranged as high as 72° F. during the day and several degrees lower during the night, but not low enough to in-

[1] Paper read before the American Phytopathological Society at the Minneapolis meeting, Dec. 29, 1910.

[2] Sheldon, J. L. Concerning the Identity of the Fungi Causing an Anthracnose of the Sweet Pea and the Bitter Rot of the Apple. Science, N. S. **22**: 51-52. 1905.

jure the plants. All the seedlings grew well and were perfectly healthy. The day before inoculation the pots with the seedlings were covered with bell jars thus forming moist chambers. These covers were removed one day after inoculation. Two methods of inoculation were employed: (1) the introduction of spores into the living stems through punctures made with a sterilized needle, (2) by liberally spraying the surface of the plants by means of an atomizer with spores suspended in sterilized water. Fruits of apples and pears on the trees in the orchard were also treated in the following manner. Healthy fruits on the trees were first washed with a 5% solution of formalin and then rinsed with distilled water. They were then inoculated through sterile needle punctures and covered with paper bags. For the inoculations, pure cultures were used of Gloeosporiums from various sources as indicated below. The results of these experiments are given in Table I.

TABLE I

| Source of Gloeosporium culture. | Number and kind of plants inoculated. | Method of inoculation. | Date of inoculation. | Results of inoculation. | Checks. |
|---|---|---|---|---|---|
| Sweet Pea | 50 sweet pea seedlings 3 weeks old | Puncture | Sept. 26 | Oct. 2, all dead | 50 all healthy |
| " " | Same | Atomizer | Oct. 26 | Nov. 7, " " | " |
| " " | Same | " | Nov. 14 | Nov. 21, " " | " |
| Apple | Same | Puncture | Oct. 10 | Oct. 18, " " | " |
| " | 50 sweet pea seedlings 4 weeks old | " | " " | Oct. 22, " " | " |
| " | Same | Atomizer | Nov. 20 | Nov. 29, 44 " | " |
| " | Same | Puncture | Oct. 26 | Nov. 1, 31 " | " |
| " | Same | " | Nov. 14 | Nov. 20, all | " |
| " | Same | Atomizer | Oct. 5 | Oct. 14, 37 " | " |
| " | 50 sweet pea seedlings 8 weeks old | " | Oct. 7 | Oct. 17, 42 " | " |
| Sweet Pea | 12 apples on tree | Puncture | Oct. 7 | Oct. 17, typical bitter rot | 5 " |
| " " | 16 " " | " | Oct. 8 | " " " | 12 " |
| Apple | 15 " " | " | " " | " " " | 16 " |
| " | 21 pears on tree | " | " " | " " " | 8 " |
| Sweet Pea | 18 " " | " | " " | " " " | 11 " |
| " " | 16 " " | " | " " | " " " | 12 " |

The data in Table I show: (1) that the original organism of the sweet pea is pathogenic to the sweet pea, (Plate XXX Fig. 1) and also to the apple in which it causes the typical bitter rot (Plate XXX Figs. 2, 3, 4); (2) that *Gloeosporium fructigenum* Berk. from the apple causes a disease on the sweet pea which is similar to the disease caused by the original sweet pea Gloeosporium. This definitely proves that *Glomerella rufomaculans* (Berk.) Spauld. & von Sch. is the cause of the anthracnose disease of the sweet pea.

TABLE II

| Species of Gloeosporium | Number and kind of plants inoculated | Method of inoculation | Date of inoculation | Results of inoculation | Check |
|---|---|---|---|---|---|
| Gloeosporium gallarum | 50 sweet pea seedlings | Puncture | Oct. 26 | Nov. 7, 42 dead | 50 all healthy |
| " | " | " | Nov. 14 | Nov. 25, 41 dead | " |
| " | " | Atomizer | Nov. 30 | Dec. 12, 8 dead | " |
| " | " | " | Dec. 2 | Dec. 14, 19 dead | " |
| " | " | " | Oct. 26 | Nov. 7, 34 dead | " |
| Gloeosporium officinale | " | Puncture | Nov. 14 | Nov. 25, 43 dead | " |
| " | " | " | Nov. 30 | Dec. 12, 50 dead | " |
| " | " | Atomizer | Dec. 2 | Dec. 14, 50 dead | " |
| " | " | " | Oct. 26 | Nov. 7, 50 dead | " |
| Species from May apple | " | Puncture | Nov. 14 | Nov. 26, 42 dead | " |
| " | " | " | Nov. 30 | Dec. 12, 17 dead | " |
| " | " | Atomizer | Dec. 2 | Dec. 14, 19 dead | " |
| " | " | " | Oct. 26 | " | " |
| Glomerella psidii | " | Puncture | " | Nov. 20, failure | " |
| " | " | Atomizer | " | " | " |
| Species from Persea | " | " | Oct. 20 | " | " |
| " | " | Puncture | " | " | " |
| Gloeosporium gallarum | 15 pears on tree | " | " | Nov. 15, typical bitter rot | 7 " |
| Gloeosporium officinale | 12 " | " | " | " | 5 " |
| Species from May apple | 14 " | " | " | " | 9 " |
| Glomerella psidii | 19 " | " | " | Nov. 15, not typical bitter rot | 6 " |
| Species from Persea | 16 " | " | Oct. 5 | " | 6 " |
| Species from sweet pea | 106 pole lima on pods | " | Oct. 7 | Oct. 19, successful | 16 " |
| Species from apple | 66 " | " | " | " | 12 " |
| Species from May apple | 24 " | " | " | " | 9 " |
| Gloeosporium officinale | 32 " | " | " | " | " |
| Gloeosporium gallarum | 42 " | " | " | " | " |
| Glomerella psidii | 60 bush | " | " | " | " |
| Species from sweet pea | 42 " | " | " | " | 19 " |
| Species from apple | 49 " | " | " | " | 12 " |
| Species from apple | 40 " | Atomizer | " | Oct. 26, failure | 12 " |
| Species from sweet pea | 58 " | " | " | " | 12 " |

*Relation of other Gloeosporiums to the sweet pea disease.* While working on the question of the identity of the bitter rot of the apple and the anthracnose of the sweet pea, it was considered desirable to determine whether other species of Gloeosporium could produce an anthracnose of the sweet pea similar to that caused by the bitter rot organism of the apple. Therefore, sweet pea seedlings were inoculated with spores from pure cultures of five different Gloeosporiums then in stock in the laboratory. The results of these experiments are given in Table II.

From Table II it will be seen that the Gloeosporium from the May apple fruit (*Podophyllum peltatum*), *G. gallarum* Ch. Rich. from oak gall, and *G. officinale* E. & E. from sassafras leaves, are able to infect sweet pea seedlings through puncture inoculations as readily as the sweet pea or the apple Gloeosporium. Furthermore, that *G. officinale* readily infects sweet pea seedlings by atomizer inoculations, the infection being nearly 100%. While *G. gallarum* and the species of Gloeosporium from the May apple fruit also infect sweet pea seedlings, the percentage of successfully inoculated seedlings is smaller with the atomizer inoculations than when the inoculations are made by needle punctures.

*Glomerella psidii* (G. Del.) Sheldon and the Gloeosporium from Persea failed to infect sweet pea seedlings.

The apple trees in the old orchard of the Experiment Station did not bear enough fruits to permit inoculation experiments with above five organisms; hence Kieffer pear trees which bore heavily were chosen for this purpose. They were accordingly inoculated with the five Gloeosporiums already mentioned. The results of these inoculation, (Table II) show that the species of Gloeosporium from the May apple *G. gallarum* and *G. officinale* produce the typical bitter rot on the pear, while the Gloeosporium from the guava and Persea infect the pear, but cause dull spots in which the acervuli are black and the spores are borne on long black conidiophores. Similar results were obtained when pear fruits were inoculated with the same Gloeosporium and kept in moist chambers in the laboratory. These experiments also show (Table II) that all the Gloeosporiums here considered, except the species from guava and Persea very readily produced an anthracnose disease on the pods of the lima beans in the field, which was similar to the anthracnose of the sweet pea, but quite unlike the bean anthracnose, *Colletotrichum lindemuthianum* (Sacc. & Mag.) B. & C. All the Gloeosporiums referred to above attacked the pods of the lima bean in the field when the inoculations were made by means of punctures, but not otherwise. The spots produced on the lima

bean pods by *Glomerella psidii* are gray with grayish acervuli and made up of black setae very similar to those of a true Colletotrichum, but unlike *C. lindemuthianum*. None of the above species of Gloeosporiums would infect bean or vetch seedlings. The same precautions were taken in inoculating the bean pods and pears in the field as with the apples.

*Morphology of the fungus.* The spores and mycelium of the Gloeosporium of the sweet pea do not differ from the corresponding structures of *Glomerella rufomaculans*. Sheldon first observed endospores of the Gloeosporium of the sweet pea in pure cultures, and the writer has

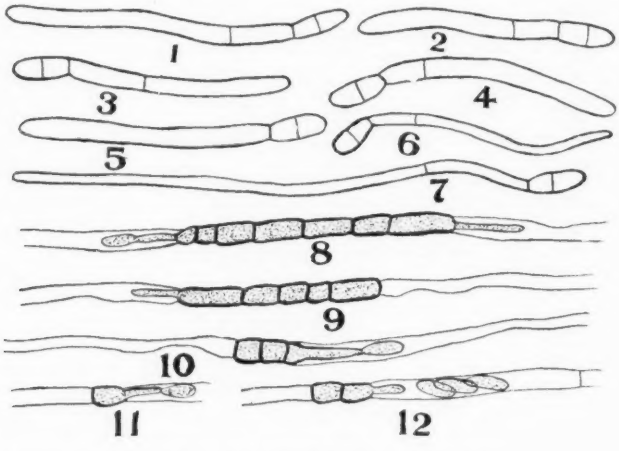



FIG. 1. Germinating spore of the sweet pea Gloeosporium.
FIG. 2. Germinating spore of the apple Gloeosporium.
FIG. 3. Germinating spore of the may apple Gloensporium.
FIG. 4. Germinating spore of *Gloeosporium officinale* E. & E.
FIG. 5. Germinating spore of *G. gallarum* Ch. Rich.
FIG. 6. Germinating spore of Gloeosporium from Persea.
FIG. 7. Germinating spore of *Glomerella psidii* (G. Del.) Sheldon.
FIG. 8. Endospore formation in *Glomerella rufomaculans* of the apple.
FIG. 9, 10, 11. Endospore formation in *Gl. psidii*.
FIG. 12. Endospore formation in Glomerella from sweet pea.

observed the same structures in hanging drop and plate cultures of *Glomerella rufomaculans* from the apple and the sweet pea, *G. officinale* and *G. psidii*.

The formation of spores is as follows. In some of the mycelial threads the protoplasmic content rounds itself into one or more cells, resembling chlamydospores. At the tip end of these cells a filament grows out within the empty part of the mycelial thread and at the tip of this filament the endospore is formed in the same manner as the

conidia on the conidiophores of a Gloeosporium. The endospore is broken off and pushed forward by the formation of a new one. (Text figs. 8-12). Further studies are necessary to determine the conditions necessary for endospore formation. The spores of *G. fructigenum*, *G. gallarum*, and the Gloesporiums from sweet pea and May apple, all germinate in the same manner, by sending out a stout germ tube (Text figs. 1-5). On five different synthetic media these Gloeosporiums produce growths and fructifications of the same character. The spores of the species from guava and Persea germinate by sending out a very thin germ tube (Text figs. 6-7). They also differ in manner of growth and fructification on the synthetic media from the other organisms used. Until the perfect stages are found, it appears from these studies that we are justified in considering *G. gallarum*, *G. officinale* and the Gloeosporium from the May apple fruit as one and the same with *Glomerella rufomaculans* (Berk.) Spaul. & von Sch. of the apple and the sweet pea.

*Life history.* In order to determine whether the disease is carried over with the seeds of the sweet pea, a large quantity of diseased pods were collected and kept over winter, some in the laboratory and some out of doors. Spores from both lots of material were tested Nov. 22, Dec. 22, 1910, Jan. 22, Feb. 22, March 15, 20, 24, April 20, May 21, and June 20, 1911. In all cases the spores germinated well and produced normal colonies on bean agar. Spores of cultures obtained from the sowing of June 20, 1911, readily infected healthy sweet pea seedlings, which were grown in pots in the laboratory. This indicates that the organism is carried over the winter as viable spores on the pods and on the seeds, and there is very little doubt that the disease is introduced into new localities through diseased seeds.

## SUMMARY

1. Through numerous cross inoculations it is definitely proven that the anthracnose disease of the sweet pea is caused by the same organism as the bitter rot of the apple, *Glomerella rufomaculans* (Berk.) Spauld. & von Sch.

2. *G. officinale* E. & E. *G. gallarum* Ch. Rich. and the *Gloeosporium* from the May apple fruit can also infect sweet pea plants, the symptoms being the same as those caused by the *Glomerella rufomaculans* of the sweet pea. The Gloeosporium from the May apple and *G. gallarum* were most successful with puncture inoculations; *G. officinale* infects sweet pea plants by atomizer inoculations as well as by puncture.

3. The above enumerated Gloeosporiums will also cause an anthracnose on lima beans and the bitter rot on pears by puncture inoculations in the field. The Gloeosporium from Persea and *Gloeosporium psidii* from guava fail to infect sweet pea plants. However they will attack the pear and the pods of lima beans but produce symptoms which are different from those produced by the other species of Gloeosporium.

4. Until perfect stages are found, the previous inoculation experiments point to the possibility that the Gloeosporium from the May apple, *G. officinale*, and *G. gallarum* are all one and the same as *Glomerella rufomaculans* (Berk.) Spauld. & von Sch. of the apple.

Material showing results of these inoculations has been deposited in the herbaria of the Delaware Experiment Station, Cornell University, the University of Wisconsin and at the National Museum of Washington, D. C.

I wish to express my thanks to Dr. Mel T. Cook and to Prof. C. A. McCue for suggestions and criticisms in reading this paper.

DELAWARE COLLEGE AGRICULTURAL EXPERIMENT STATION,
NEWARK, DELAWARE

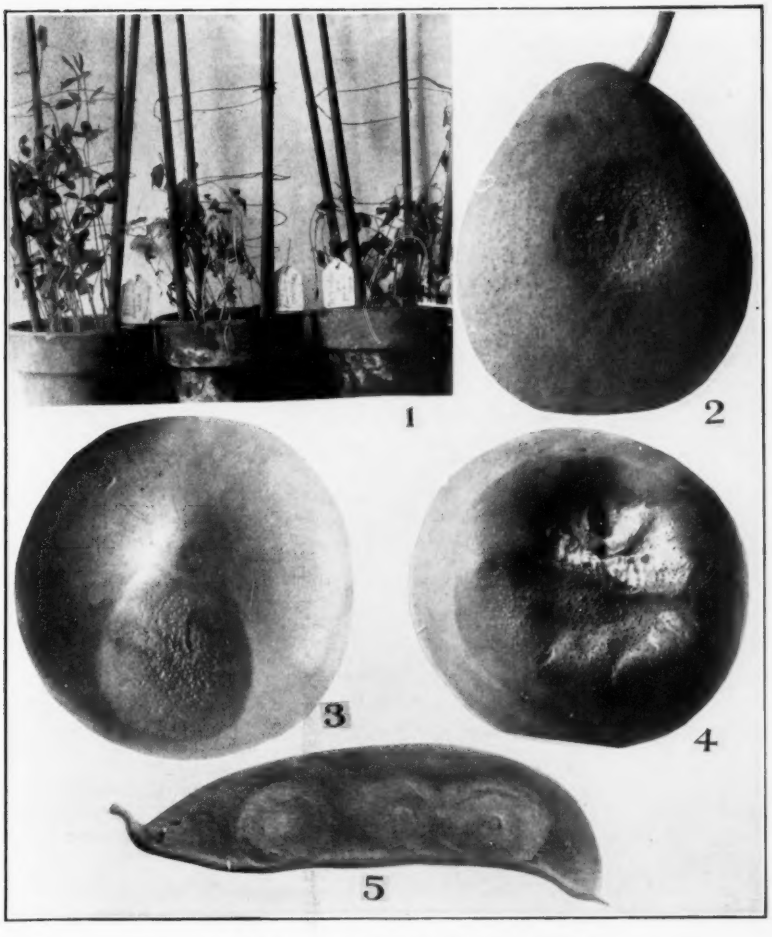



PLATE XXX. **Glomerella rufomaculans**

FIG. 1. Sweet pea seedlings inoculated with *Glomerella rufomaculans* (Berk.) Spauld. & von Sch. of the apple.

FIG. 2. Pear inoculated on the tree with *Glomerella rufomaculans* of the sweet pea.

FIG. 3. Early stage of bitter rot of apple produced by *Glomerella rufomaculans* of the sweet pea.

FIG. 4. Later stage of the bitter rot of apple produced by *Glomerella rufomaculans* of the sweet pea.

FIG. 5. Lima bean pods inoculated with *Glomerella rufomaculans* of the sweet pea.

# REVIEWS

Smith, Erwin F. Bacteria in relation to plant diseases. Vol. 2. History, general consideration, vascular diseases. Quarto, 368 pages, 22 plates (4 colored), 149 text-figures. 1911. Published by the Carnegie Institution of Washington, D. C. Price $5.00.

The second volume of Dr. Smith's exhaustive work, has appeared under date of October 30th. Both the figures and the text bear constant witness to the truth of the author's statement that the work is based "in great part on the data obtained as the result of a multitude of experiments made by the writer and his assistants, but all sources of published information have been considered." Plant pathologists are indeed under deep obligations both to the author who has so persistently devoted himself to this stupendous work and to the institution which has aided in it. This volume really marks the beginning of the detailed treatment of bacterial diseases of plants, volume I having dealt primarily with methodology. It is of the greater interest because it takes up at the outset certain fundamental questions.

The contents of these can only be touched upon. First comes a detailed review of the history of knowledge of bacteria in relation to plant diseases. Due credit is given to the pioneer work of the American, Burrill (1878), on pear-blight, the Frenchman, Prillieux (1879), on the rose-red disease of wheat, the Hollander, Wakker (1883), on the yellow disease of hyacinths, and the Italian, Comes (1880), on various diseases. The author after pointing out the sanely conservative attitude of DeBary (1888) does not fail to pay his compliments "lest we forget" to the ignorance and unscientific attitude of certain later bacteriologists, especially Alfred Fischer (1897), in denying the possibility of bacterial diseases of plants.

The most interesting parts of the volume to the general biologist, and scarcely less so to the pathologist, are found in the chapter on the general relations of bacteria to plant tissues. The subjects discussed include the normal freedom of healthy plant tissues from bacteria, the mode of entrance of bacteria into plants, what constitutes a parasite, the carriers of infection, the inception and progress of disease, symbiosis, and the possibilities of interrelations between bacterial diseases of animals and of plants. Critical discussion, where the opinions of the author are of especial interest, follows on the relations of the bacteria to the plant tissues. Emphasis is properly laid on the mass action of bacteria when it comes to tissue invasion and there is justifiable skepticism as to the migration through cell walls of isolated organisms. There is a detailed account of the knowledge to date of the action of bacteria on cell walls, the solution of middle lamellae, of cellulose walls, and the destruction of wood. The reaction of the host plant cells is also considered in detail. In hyperplasia the multiplication of cells is attributed to repeated cell division under the stimulus of microorganisms within the cells. "Just what this stimulus is we do not know. It is probably a definite chemical substance, an endotoxin. The writer suspects ammonium or calcium oxalate to be one of the stimulating substances." Some interesting suggestions follow relative to hypertrophies, especially tyloses, the enlargement of nuclei, the changes of chromosomes, and the possibilities of acquired resistance and antibodies.

The consideration of secondary tumors and metastasis as these occur in the olive tubercle and daisy gall diseases is illustrated by some remarkably interesting figures. These secondary galls may develop long distances from the primary infection, even two or three feet in the olive. In this host there is traceable a distinct channel of infection from the primary to the secondary tubercle, with disorganized tissue and demonstrable bacteria. In the daisy "a strand of tumor tissue has been discovered connecting the primary tumor with the secondary tumors." The bacteria are here believed to occur only intracellularly, not only

in the primary and secondary tumors, but, sparingly, in the cells of the connecting strand. The interesting theory is held that the development of these tumor tissues depends upon a very delicate series of adjustments between the attacking organisms and the host cells, which the author explains as a hitherto unrecognized type of bacterial relation, and he points out that the phenomena "throw a flood of light on the mechanism of the development of malignant animal tumors." If this be accepted, as the facts outlined seem to justify, we have the satisfaction of seeing a notable repayment of the debt plant pathologists have heretofore owed to animal pathologists for methods and theories.

The critical and detailed review of the status of knowledge as to root nodules of Leguminosae and symbiosis constitutes an unexpected, but welcome, contribution, based upon bibliographical lists of some 300 titles.

The discussion of specific diseases is then taken up, the basis of classification proposed being (1) vascular diseases, (2) parenchyma diseases without hyperplasia, (3) cankers, tubercles, and tumors with hyperplasia. Wilt of cucurbits is discussed first and in greatest detail; 90 pages with some 50 figures and plates, some of which are from originals made as early as 1893. Any attempt at detailed review is prohibited by space limitations. This part will be especially welcome to plant pathologists of the northern United States, owing to the widespread destructiveness of this disease and the absence heretofore of any single comprehensive publication. The treatments of black rot of cruciferous plants and yellow disease of hyacinths, each filled with illustrations, bring the reader to the close of the volume with a keen anticipation of what the succeeding volume may contain and the earnest wish that it soon be forthcoming. Certainly nothing comparable to this as fundamental work in plant pathology has appeared in this generation.

L. R. JONES.

## PHYTOPATHOLOGICAL EXHIBITION

### NOTICE

Plans have been made for a Phytopathological Exhibit at the forthcoming meeting of the Society in Washington, D. C., December 27-30. Members and others interested in the subject are hereby sequested to cooperate in making an exhibit which shall creditably reflect the present attainments of phytopathology. The exhibits will be placed in a room adjoining the hall in which the meetings of the society are held.

The following materials are suggested for the purpose:

(a) Photographs suitably mounted; (b) Specimens of diseased material, dried fungi, etc.; (c) Pure cultures of pathogenic organisms; (d) Instruments, special apparatus and other appliances useful in research or in teaching the subject; (e) Microscopic preparations.

All material should be neatly and adequately labeled and should bear the name of the person or laboratory exhibiting the same.

The Council of the Society has placed Dr. H. S. Reed, Blacksburg, Va., in charge of the exhibit feature of the meeting. Exhibitors are requested to communicate to him as early as possible the character of their exhibits and the approximate amount of space required. The number of microscopes available is limited, consequently those requiring microscopes should write early for reservations. Those who cannot be present in person may send their exhibits to

H. S. REED, Dept. Agriculture,
Care of C. L. Shear. Washington, D. C.

# INDEX TO VOLUME I

**New scientific names are printed in full face type**


Aecidium trifolii-repentis, 3, 6
Alfalfa rust, 108
Althea rosea, 57
Anthracnose, a study of, 106; bean, varietal resistance to, 107; cotton, 108; Schizanthus, 120
Apple, new fruit spot, 32, 108; hairy root, 10; water-core, 128
Apple trees, effect of arsenicals, 79
Armillaria mellea, 104
Autoecism, 104

Bacillus amylovorus, 156, 157; persistence of in apple twigs, 68; — carotovorus, 76; — coli, 97, 98, 99; a plant parasite, 97; — musae, 48; — tumefaciens, 106; — solanacearum, 48; — vitivorus, 116
Bacteria in relation to plant diseases,
Bacterial disease of banana and plantain, 45, 108
Bacterium tumefaciens, 7, 8, 106
Banana, bacterial disease of, 45, 108
Barre, H. W., Results of cotton anthracnose work for 1910, 108
Barrus, M. F., Varietal resistance of beans to anthracnose, 107
de Bary, Anton, biographical sketch of, 1
Baumkrankheiten, Versuche über, 105
Beans, varietal resistance to anthracnose, 107, 190
Beckwith, T. D., Root and culm infections by soil fungi in North Dakota, 169
Beet, leaf spot of, 108
Bjerkandera, 179
Black rot disease of the grape, 167
Blaufäule des Nadelholzes, 104
Burger, O. F., Fawcett, H. S. and, 164

Cabbage, black leg, 28; club root of, 159; Fusarium wilt, 28; Phoma wilt, 28
Caeoma abietis-canadensis, 94; tsugae, 95
Calamagrostis canadensis, Sclerotium rhizoides on, 69
Cankers of peach trees, 66
Carya tomentosa, 134
Castanopsis chrysophylla, 132
Cenangium abietis, injury to Pinus strobus, 180
Cenangium ferruginosum, 183
Cephalothecium, 171
Ceratostomella pilifera, 104
Cereal smut spore germination, 108
Chestnut, bark diseases, 106
Citrus, scaly bark, 164; aurantium, 65, 164; Cladosporium on, 164; hystrix, var. acida, 63, 65
Cladosporium graminum, 19, 20, 23, 26; herbarum on citrus, 164; herbarum var. **citricolum,** 165
Claviceps, method of developing, 50; purpurea, 50, 51
Clover, red, rust of, 3; white, rust of, 3
Club root of cabbage, 159
Colletotrichum, on cotton, 169; on wheat, 169
Colletotrichum lindemuthianum, strains of 194; **schizanthi,** 125
Collybia velutipes, 104, 105
Coniothyrium fuckelii, parasitism of, 100
Cook, M. T., Tannin in relation to the enzymes of the plant cells, 68; Notes on hypertrophied structures, 108
Cook, Mel T., and Taubenhaus, J. J., Trichoderma Köningi the cause of a disease of sweet potatoes, 184
Coniophora cerebella, 104, 108
Corticium laetum, 16, 17; roseum, 17
Cotton anthracnose, 108
Cronartium asclepiadeum, 146; quercuum, 131, 132
Crown gall of grape, 106; of plants, 7, 108
Crown swelling disease of peach, 53
Cryptospora aesculi, 118; aurea, 118; compta, 118; daldiniana, 118; ehrenbergii, 118; foraminula, 118; hypodermia, 118
Cryptosporella, 106; veneta, 118; **viticola,** 119
Cylindrosporium pomi, 34
Cystopus, spore germination, 69
Cystopus candidus, 75

Dactylis glomerata, 50
Dasyscypha wilkommii, 105
Dendrophagus, 10
Diseases of economic plants, 35
Diseases of trees, 108
Distichlis spicata, 147
Dolichos sesquipedalis, 191
Duggar, B. M., Physiological plant pathology, 71
Durand, E. J., Differential staining of intercellular mycelium, 129

Edgerton, C. W., Two new fig diseases, 12
Euphorbia commutata, 5
Exoascus deformans, 107

Fagus sylvatica, 105
Fawcett, H. S., and Burger, O. F., A variety of Cladosporium herbarum on Citrus aurantium in Florida, 164
Festuca elatior, 50
Ficus carica, 16
Fig, two new diseases, 12; canker, 12; limb blight, 16
Fink, Bruce, Injury to Pinus strobus caused by Cenangium abietis, 180
Fomes annosus, 104, 108; fomentarius, 104, 105; fulvus, 104; igniarius, 104, 105; pinicola, 104
Forest tree diseases, 108
Freeman, E. M., Resistance and immunity in plant diseases, 109
Fruit spot of apple, 32, 108
Fruit trees, silver leaf, 177
Fulton, H. R., The persistence of Bacillus amylovorus in pruned apple twigs, 68
Fungi, mine, 108
Fungicides, studies on, 107
Fusarium, blight and rot of potato, 70; scab of wheat, 169; wilt of cabbage, 28; wilt of flax, 169
Fusicladium effusum, 133
Fusicoccum viticolum, 106, 116, 118, 119

Gloeosporium fructigenum, 106; gallarum, 106; guava, 106; officinale, 106; persea, 106; psidii, 199, 200
Gloeosporiums, relation to a sweet pea disease, 196
Glomerella rufo-maculans, 106
Grand Rapids tomato disease, 108
Grape, black rot disease of, 167; dead arm, 106, 116
Guignardia bidwellii, 167
Güssow, H. T., Preliminary note on silver leaf disease of fruit trees, 177
Gymnosporangium clavariaeforme, 147; globosum, 147

Hairy root, apple, 10
Hall, J. G., Stevens, F. L. and, 35
Hawkins, L. A., Review by, 168
Heald, F. D., Rhizoctonia medicaginis in America, 103
Hedgcock, G. G., Notes on Peridermium cerebrum Peck, and Peridermium harknessii Moore, 131; Notes on some diseases of trees in our natural forests, 108
Hedges, Florence, Sphaeropsis tumefaciens, n. sp., the cause of the lime and orange knot, 63
Helminthosporium on barley, 169
Heteroecism in rusts, 139
Humphrey, C. J., A contribution to our knowledge of mine fungi, 108
Hypertrophy, notes on, 108
Hypholoma fascicularis, 104

Immunität, Untersuchungen über, etc., 104

Jensen, C. N., and Stewart, V. B., Anthracnose of Schizanthus, 120
Johnson, E. C., Floret sterility of wheats in the southwest, 18
Jones, D. H., Scolytus rugulosus as an agent in the spread of bacterial blight in pear trees, 155
Jones, L. R., The relations of plant pathology to the other branches of botanical science, 39; review by, 203
Johnston, J. R., Is Bacillus coli ever a plant parasite? 97

Kern, F. D., The rusts of white and red clover, 3

Lavatera plebeia, 57
Lenzites betulina, 104, 105, 108; sepiaria, 105
Lime, knot, 63
Lolium temulentum, 113
Leaf blotch, pecan, 133
Leaf spot of sugar beets, 108

Macrosporium, 171
Malva crispa, 57, 61, rotundifolia, 56, 57, 58, 59, 60, 61; sylvestris, 57
Manns, T. F., Black-leg or Phoma wilt of cabbage, 28; The Fusarium blight and rot of potato, 70
Marasmius, 49
Merulius lacrymans, 104, 108
Melampsorella caryophyllacearum, 147
Melhus, I. E., The relation of temperature to spore germination and infection with Cystopus, 69
Metcalf, H., and Collins, J. F., Further notes on the bark diseases of the chestnut, 106
Mine fungi, 108
Morris, H. E., Swingle, D. B and, 79
Münch, E., Die Blaufäule des Nadelholzes, 104
Münch, E., Untersuchungen über Immunität, etc., 104
Münch, E., Versuche über Baumkrankheiten, 105
Mycelium, The differential staining of intercellular, 129
Mycosphaerella 133; convexula, 135
Musa chinensis, 46; paradisiaca, 46; textilis, 47

Necium farlowii, 94
Nectria cinnabarina, 105; ditissima, 105
Necrosis, grape, 106, 116
Norton J. B. S., Crown swelling disease of peach, 53; Water core of apple, 126
Nursery stock, Peach leaf-curl, 107

O'Gara, P. J., Parasitism of Coniothyrium fuckelii, 100

Olive, E. W., Origin of heteroecism in the rusts, 139
Orange knot, 63

Paxillus panuoides, 108
Pea, anthracnose of, 106
Peach, crown swelling disease of, 53; leaf curl, 107; winter killing of, 66
Pear, bacterial blight, 155
Pecan, leaf-blotch, 133
Peridermium cerebrum, 131, 132; fructigenum, 95, 96; harknessii, 131, 132; peckii, 94
Peronospora parasitica, 129
Phalaris arundinacea, 140
Phaseolus lunatus, 191; vulgaris, susceptibility, 191
Pholiota squarrosa, 104; adiposus, 104
Phoma brassicae, 28; oleracea, 29, 30
Phytophthora infestans, 75, 77
Pinus divaricata, 131; echinata, 131; murrayana, 132; ponderosa, 132; radiata, 132; rigida, 132; strobus, 131; sylvestris, 131, 132, 146; virginiana, 131
Pinus strobus, injury to by Cenangium abietis, 180
Plantains, bacterial disease of, 45, 108
Plant Diseases, resistance and immunity, 109
Plant pathology, industrial fellowship in, 68; physiological, 71; relations of to botanical science, 39
Plasmodiophora brassicae, 10, 159
Plaginathus spicatus, 57
Podophyllum peltatum, 106
Polystictus cinnabarina, 105; hirsutus, 105; pergamenus, 105; versicolor, 105, 108
Populus tremula, 118
Potato, Fusarium blight, 70; rot, 70
Pritchard, F. J., The wintering of Puccinia graminis tritici and the infection of wheat through the seed, 150
Puccinia caricis, 147; coronata, 141; coronifera, 141, 147; glumarum, 113; graminella, 141; graminis, 110, 140, 143, 145, 146, 152, 153; graminis tritici, 20, 22, 23, 24, 25, 26, 150; wintering of, 150;—malvacearum, morphology and life history of, 55; malvacearum, 55, 61;—mesnieriana, 141; rubigo-vera tritici, 20; trifolii, 5, 6; trifolii repentis, 5; trifolii filiformis, 5; trifolii hybridi, 5; subnitens, 147
Pucciniastrum minimum, 96
Pythium deBaryanum, 75

Quercus alba, 131; californica, 131; coccinea, 132; densiflora, 131; var. echinoides, 131; emoryii, 131; gambellii, 131; lobata, 131; marilandica, 131; Michauxii, 132; minor, 132; phellos, 132; prinus, 132; rubra, 131; texana, 132; undulata, 132; velutina, 132; virginiana, 132

Rand, F. V., A pecan leaf blotch, 133
Reddick, Donald, Necrosis and crown gall diseases of grapes in New York, 106; and Whetzel, H. H., 50
Reed, H S., The effect of the club root disease upon the ash constituents of the cabbage root, 159
Resistance and immunity in plant diseases, 109
Rhizoctonia medicaginis in America, 103
Rhizoctonia violacea, 103
Rolfs, F. M., Winter-killing of twigs, cankers and sun-scald of peach trees, 66
Root and culm infections of wheat by soil fungi, 169
Rorer, J. B., A bacterial disease of bananas and plantains, 45
Rust, alfalfa, 108; clover, red and white, 3; origin of heteroecism in, 139; timothy, 108

Schizanthus, anthracnose of, 120
Schizophyllum commune, 104, 105
Sclerotium rhizoides on Calamagrostis, 69
Scolytus rugulosus, spread of pear blight by, 155, 156, 157
Scott, W. M., A new fruit spot of apple, 32
Secale cereale, 50
Shear, C. L., The ascogenous form of the fungus causing dead-arm of the grape, 116. Reviews by, 35, 66
Silver leaf disease, of fruit trees, 177
Smith, Erwin F., Anton deBary, 1; Crown gall of plants, 7, 108; The Grand Rapids tomato diseases, 108
Smut spore germination, 108
Soil fungi, root and culm infection of wheat by, 169
Spaulding, P., The white pine blister rust, 70; The rusts of Tsuga canadensis, 94; —Review by, 104
Sphaerella convexula, 134, 135
Sphaeria convexula, 135; limminghii, 118
**Sphaeropsis tumefaciens**, 64
Sphaeropsis tumefaciens, nov. sp., the cause of the lime and orange knot, 63
Spore germination, relation of temperature to, with Cystopus, 69
Staining of intercellular mycelium, 129
Stakman, E. C., Cereal smut spore germination, 108
Stemphylium tritici, 20, 22, 23, 26, 27
Stereum fasciatum, 108; hirsutum, 104, 105; purpureum, 104, 105; rugosum, 105

Stevens, F. L., and Hall, J. G., Diseases of economic plants, 35
Stewart, V. B., Peach leaf curl in nursery stock, 107; Jensen, C. N., and, 120
Stout, A. B., Sclerotium rhizoides on Calamagrostis canadensis, 69
Sugar beet, leaf spot, 108
Sun scald of peach trees, 66
Sweet pea disease, 106
Sweet potato disease, 196
Swingle, D. B., and Morris, H. E., A preliminary report on the effects of arsenical compounds upon apple trees, 79, 108

Tannin, Relation to enzymes, 68
Taubenhaus, J. J., A contribution to our knowledge of the morphology and life history of Puccinia malvacearum Mont., 55; A study of some Gloeosporiums and their relation to a sweet pea disease, 106, 196; Cook, M. T. and, 184
Timothy rust, 108
Tomato disease, Grand Rapids, 108
Townsend, C. O., Leaf spot of sugar beets, 108
Trichobasis fallens, 6
Trichoderma köningi, 185; —lignorum, 184.
Trifolium carolinianum, 5; hybridum, 3, 4, 6; incarnatum, 3, 4, 6; medium, 6; pratense, 3, 4, 6; repens, 3, 4
Triticum monococcum, 77
Tsuga canadensis, 96; rust of, 94; caroliniana, 94
**Tubercularia fici,** 13, 16

Uredo apiculata, 6; a trifolii, 6; apiculosa var. trifolii, 6; fabae trifolii, 6; fallens, 5, 6; trifolii, 6
Uromyces elegans, 5; **fallens**, 6; pisi, 147; trifolii, 3, 4, 5, 6; trifolii-repentis, 3, 6
Ustilago avenae, 110

Valsa leucostoma, 66, 105; var. cincta, 66; var. rubescens, 66; sordida, 105
Vicia faba, 191

Wallace, E., Blodgett, F. M., and Hesler, L. R., Some laboratory and field studies on fungicidal values, 107
Water core of apple 126
Wheat, floret sterility, 18; infection through seed, 150; root and culm infection, 169
Whetzel, H. H., The industrial fellowship in plant pathology, 68
Whetzel, H. H., and Reddick, D., A method of developing Claviceps, 50
White pine blister rust, 70
Winter killing of peach trees, 66

Vol. I No. 6

# Phytopathology

Official Organ of the

American Phytopathological Society

DECEMBER, 1911

CONTENTS

Root and Culm Infections of Wheat by Soil Fungi in North Dakota ..... T. O. BIDGWITH 163

Preliminary Notes on Silver Leaf Disease of Fruit Trees ..... H. T. GUSSOW 177

Injury to Pinus Strobus caused by Cenangium Abietis ..... BRUCE FINK 180

Trichoderma Koningi the Cause of a Disease of Sweet Potatoes ..... MEL. T. COOK AND J. J. TAUBENHAUS 183

Variation of Varieties of Beans in their Susceptibility to Anthracnose ..... MORTIER P. BARRUS 200

A Study of Some Glomerellaceae and their Relation to a Sweet Pea Disease ..... J. J. TAUBENHAUS 205

Reviews ..... E. F. SMITH 213

Phytopathological Exhibition ..... 224

Entered as second-class matter at the post-office at Ithaca, N. Y., under the Act of March 3, 1879.

Andrus & Church, Printers, Ithaca, N. Y.

# PHYTOPATHOLOGY

THE OFFICIAL ORGAN OF

THE AMERICAN PHYTOPATHOLOGICAL SOCIETY

*EDITORS*

L. R. JONES,
University of Wisconsin, Madison, Wis.

C. L. SHEAR,
U. S. Department of Agriculture,
Washington, D. C.

H. H. WHETZEL,
Cornell University, Ithaca, N. Y.

*ASSOCIATE EDITORS*

G. P. CLINTON, Agricultural Experiment Station, New Haven, Conn.
E. M. FREEMAN, University of Minnesota, College of Agriculture, St. Paul, Minn.
H. T. GÜSSOW, Central Experimental Farm, Ottawa, Canada.
F. D. HEALD, University of Texas, Austin, Texas.
HAVEN METCALF, U. S. Department of Agriculture, Washington, D. C.
W. A. ORTON, U. S. Department of Agriculture, Washington, D. C.
W. M. SCOTT, U. S. Department of Agriculture, Washington, D. C.
A. D. SELBY, Agricultural Experiment Station, Wooster, Ohio.
ERWIN F. SMITH, U. S. Department of Agriculture, Washington, D. C.
RALPH E. SMITH, Southern Cal. Path. Lab. and Expt. Station, Whittier, Cal.
F. L. STEVENS, College of Agriculture and Mechanic Arts, W. Raleigh, N. C.
ROLAND THAXTER, Harvard University, Cambridge, Mass.

*BUSINESS MANAGER*

DONALD REDDICK, Cornell University, Ithaca, New York.

PHYTOPATHOLOGY is published bimonthly for the Society by the Business Manager.

*EDITORIAL NOTICES*

*Manuscript for Publication* or correspondence relative thereto may be addressed to L. R. Jones, or to any other member of the Editorial Board. Contributors are requested, so far as convenient, to carry on preliminary correspondence and submit copy of manuscript to the nearest Associate Editor. For instance, correspondence from Canada may be addressed to H. T. Güssow, from New England to G. P. Clinton and from the Pacific states to Ralph E. Smith.

Manuscripts should be typewritten and carefully revised before submission. While no attempt will be made at exact uniformity in capitalization, punctuation, citations, footnotes, etc., each contributor is requested to give careful attention to clearness and preciseness in these matters and to conform as far as practicable to the best usage in this journal.

*Illustrations.* Half-tone plates and zinc etchings will be used for illustrations. For such purposes good photographs or ink drawings are best, usually of such size that they may be reduced somewhat in reproduction. About four plates may be inserted per number, or one plate for nine printed pages. If more than this is required, the author may be requested to meet the expense of extra plates or else the publication may be delayed until favorable time for its insertion.

*Separates.* Twenty-five unbound separates will be sent, gratis, to each contributor. Additional separates and covers are supplied at cost when ordered in advance. A table showing cost, with an order slip, is sent with proof.

*Reviews.* Publications for review should be sent to C. L. Shear, U. S. Dept. Agriculture, Washington, D. C.

*BUSINESS NOTICES*

*Subscription price*, $3.00 per year for all points within the United States; for foreign orders the charge is $3.25; single numbers 60 cents.

*Advertising rates* furnished on application.

*Requests to supply lost copies* of the journal must be made within 30 days from date of issue. Members should notify promptly the Secretary-Treasurer and Business Manager, Subscribers the latter, of any change of address.

*Business correspondence* and subscriptions should be sent to

DONALD REDDICK, Business Manager, Ithaca, N. Y.

# The Rex Companies

THE ONLY EXCLUSIVE MANUFACTURERS OF CLEAR,
UNIFORM, DEPENDABLE, CONCENTRATED
LIME AND SULPHUR SOLUTION
IN THE UNITED STATES
OR CANADA

---

The manufacture of Lime and Sulphur Sheep and Cattle Dip and Lime and Sulphur Spray Solution as a commercial proposition originated with us. The claims made by manufacturers of chemicals that the making of Lime and Sulphur Solution by them is a side issue is a correct statement.

Our manufacturing plants, ***where nothing but Lime and Sulphur Solution is made,*** are located at

| | | |
|---|---|---|
| Omaha, Nebr. (Home Office) | Toledo, Ohio | N. Yakima, Wash. |
| Rochester, N. Y. | Benicia, Calif. | Wenatchee, Wash. |
| | Payette, Idaho | |

REX Companies that have just organized and will operate plants this coming season will be found at

Walla Walla, Wash., and Brighton, Ontario, Can.

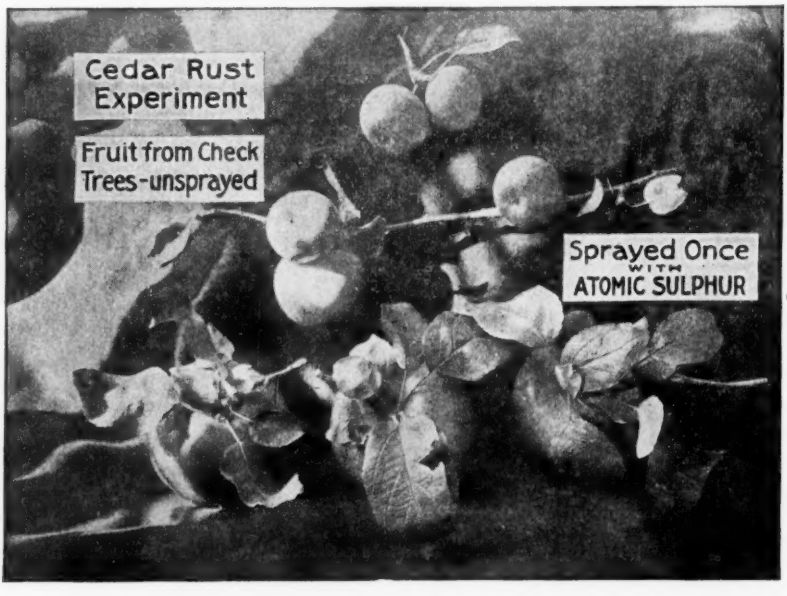
We would be pleased to have the merits of REX tested at our expense. Solution for any experimental or test work will be furnished without expense by addressing

## THE REX COMPANY,

***P. O. Box 712.*** ***Rochester, N. Y.***

# ATOMIC SULPHUR
—A—
# FUNGICIDE

Pure Sulphur in the form of a Paste




*"Orchard Brand"*

Lime-Sulphur Solution Soluble Oil

*Arsenate of Lead*

**Bordeaux Mixture** **Bordeaux Arsenate Lead Mixture**

*Sulphur* *Blue Stone*

Technical and C. P. Chemicals

MANUFACTURED BY

**THOMSEN CHEMICAL CO.**

BALTIMORE, MD.

Arsenate of Lead

Lime Sulphur Solution

## Grasselli Arsenate of Lead

A reliable insecticide. Successfully used in all the large fruit growing sections.

## Grasselli Lime Sulphur Solution

For control of San Jose Scale, Oyster Shell Scale, and all scale or sucking insects.

Also an efficient Fungicide.

It has been found especially satisfactory as a substitute for Bordeaux Mixture to control Fungous Diseases of Apples and Pears.

## Grasselli Bordeaux Mixture Paste, Powdered

A reliable fungicide.

## Grasselli Bordeaux Lead Arsenate Mixture Paste, Powdered

A combined fungicide and insecticide.

We can also furnish a high grade Copper Sulphate (Blue Vitriol).

## Grasselli Spray Products

Are guaranteed to comply with the Insecticide Act of 1910.

## *The Grasselli Chemical Company*

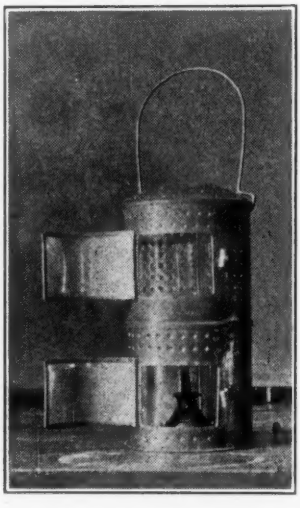
Established 1839

***General Offices, The Arcade, Cleveland, Ohio***

BRANCH OFFICES

New York—60 Wall Street.
Boston, Mass.—655 Summer Street.
Chicago, Ill.—2235 Union Court.
Cincinnati, Ohio—Pearl and Eggleston Streets.
St. Louis, Mo.—112 Ferry Street.
Milwaukee, Wis.—Canal & 16th Sts.
St. Paul, Minn.—172 & 174 East 5th St.
Detroit, Mich.—Atwater and Randolph Streets.
Birmingham, Ala.—825 Woodward Building.
New Orleans, La.—Godchaux Building.

*Send inquiries and orders to nearest Office.*



# This Kills Mildew

## EVERY TIME

FOR PARTICULARS ADDRESS

## *Benjamin Dorrance*

***ROSE GROWER***

*3 Dorrance Farm* *Dorranceton, Penn'a*

# AMERICAN PHYTOPATHOLOGICAL SOCIETY

## OFFICERS

1911

President, A. D. SELBY,
Botanist of the Ohio Agricultural Experiment Station, Wooster, Ohio.

Vice-President, R. A. HARPER,
Professor of Botany, University of Wisconsin, Madison, Wis.

Secretary-Treasurer, C. L. SHEAR,
Pathologist, Bureau of Plant Industry, Washington, D. C.

---

## ADDITIONAL MEMBERS OF THE COUNCIL

ERWIN F. SMITH,
Pathologist, Bureau of Plant Industry, U. S. Department of Agriculture, Washington, D. C.

G. P. CLINTON,
Botanist, Agricultural Experiment Station, New Haven, Conn.

F. L. STEVENS (*ex-officio*),
Professor of Botany and Vegetable Pathology, North Carolina College of Agriculture and Mechanic Arts and Biologist of the North Carolina Agricultural Experiment Station, West Raleigh, N. C.

L. R. JONES (*ex-officio*),
Professor of Plant Pathology, University of Wisconsin, Madison, Wis.